

The association between Deciduous Molar Hypomineralization and Molar Incisor Hypomineralization – report of a case

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Abstract : Developmental defects in both deciduous and permanent dentitions occur as a result of disturbances in formation of hard tissue matrices and in their mineralization. The simultaneous development period of first permanent molars and the second deciduous molars is suggestive of a shared etiological factor in the origin of hypomineralization defects. Children with hypomineralized second deciduous molars are more susceptible to develop molar incisor hypomineralization. Therefore, the purpose of this case report is to document the association of deciduous molar hypomineralization with molar incisor hypomineralization in a 8-year old male child. This report further highlights the rise in demand for more attention at the age of 6-8 yrs, the period of the eruption of first permanent molars and permanent incisors.

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I. Introduction:

Developmental defects of enamel occur as a result of disturbances in formation of hard tissue matrices and in their mineralization during amelogenesis.¹ These defects are broadly divided into two distinct categories: a) enamel hypoplasia b) enamel hypomineralization.

Enamel hypoplasia occurs due to disturbances of ameloblasts in the secretory phase of amelogenesis, which is a quantitative defect of enamel.² Clinically, hypoplastic defect presents as a symmetrical lesion of reduced enamel thickness with a smooth outline between the affected and normal tooth structure.

Hypomineralized enamel is defined as a qualitative defect resulting from the disruptions in the transitory ameloblasts during the early maturation phase. Hypomineralization defects are clinically identified as sharply demarcated, asymmetrical opaque lesions varying from white to yellow or brown in colour. Post-eruptive enamel breakdown occurs from masticatory forces that are exerted on the soft and porous hypomineralized enamel that may result in total absence of enamel in severe cases. Hypomineralized molars are more prone to post eruptive breakdown of enamel as compared to incisors when it occurs because of masticatory forces.³

A wide variety of terminologies have been previously reported in the dental literature for almost identical developmental defects of enamel in molars such as hypomineralised first permanent molars(FPM)², idiopathic enamel hypomineralization in FPM⁴, non-fluoride hypomineralization in FPM⁵ and cheese molars⁶. To avoid confusion and to better understand the concept of hypomineralization defects in a univocal manner, the use of appropriate terminology is highly recommended.⁷

In 2001 the widely accepted term, Molar-Incisor Hypomineralization(MIH) was introduced and it was defined as the hypomineralization of systemic origin that affects one to four first permanent molars, frequently associated with affected permanent incisors.⁷

Post eruptive breakdown in the MIH affected tooth may cause atypical cavitation and enamel disintegration with irregular margins on the occlusal surface. This defect is progressive and causes tooth hypersensitivity, pain during restorative treatments, atypical fillings and in severe cases, extraction of the affected teeth is indicated.^{3,5}

MIH-like defects can also occur on second deciduous molars, permanent cuspids and second molars.² Hypomineralization defects in the deciduous second molars are termed as Deciduous Molar Hypomineralization (DMH).⁸ An association between the prevalence of DMH and MIH in 5- to 6-year-old children has been reported.⁹ This relationship suggests that DMH can be used as a predictor for MIH. The purpose of this report is to present a clinical case of DMH & its association with MIH.

Case report:

A 8-year-old male child reported to the Department of Pediatric and Preventive Dentistry, Maharishi Markandeshwar College of Dental Sciences and Research, Mullana, Haryana, India with a chief complaint of decayed teeth in upper and lower back tooth region.



Figure 1) Intra-oral view of mandibular arch showing hypomineralized first permanent molars and second deciduous molar of both sides



Figure 2) Intra-oral view of maxillary arch showing hypomineralized first permanent molars and second deciduous molars of both sides

During intraoral examination, enamel hypomineralization was identified in the all first permanent molars and second deciduous molars. The patient complained of intermittent pain in second deciduous molars and permanent 1st molars. On clinical examination, dental caries with post eruptive enamel breakdown and atypical large cavities covering more than one tubercle were recorded. Deciduous first molars were found to be decayed. On radiographic examination, 2nd deciduous molars and permanent 1st molars were affected by deep carious lesions along with yellowish brown demarcated opacities and posteruptive enamel breakdown (Figure1&2). The scoring & severity of hypomineralization were recorded clinically (Table 1).

Table 1. Depicting the relationship between DMH and MIH based on scoring(EAPD criteria, 2003) ¹⁰ and severity criteria(Alaluusua et al, 1996) ¹¹

Teeth affected by hypomineralization	Deciduous 2 nd molars	Permanent mandibular 1 st molars	Permanent maxillary 1 st molars
Scoring	2	2(right side) 3(left side)	2
Severity	Moderate	Severe	Severe

II. Discussion:

This paper highlights the association between DMH and MIH, indicating that DMH has a positive correlation with the development of MIH. The development of first permanent molar starts during the 28th week of intrauterine life whereas second deciduous molar starts its development earlier to first permanent molar. Hypomineralization defects occurs due the disturbance in the maturation phase of amelogenesis.

In the deciduous second molars, the first sign of mineralization is seen around 5th month of gestation and the cusps become united at birth followed by complete coronal calcification at the age of 11-12 months after birth. However, in the permanent first molar the first evidence of calcification is seen at or around birth at the

cuspid tips that get united at the age of 6 months. But unlike the 2nd deciduous molars, its maturation phase has a longer period of 2-3 years.¹³ The coinciding maturation phase of one year in the development of second primary and the first permanent molar is considered as the period of developmental overlap.

This critical overlapping period of the enamel maturation and the relative position of the second deciduous molar & first permanent molar within the maxilla and mandible may be suggestive of a common etiological factor in the development of hypomineralization defects in both deciduous and permanent dentitions.¹⁴

In an attempt to reveal etiological factors and the putative association of hypomineralization in both primary and permanent dentitions, considerable number of genetic and environmental factors have been implicated as contributors to MIH.¹⁵ Environmental factors include illness occurring during overlapping period of deciduous second molars, FPMs and permanent incisors (perinatally and postnatally during the first three years of life) such as repeated episodes of high fever with more than 5 episodes during the first year of life, chicken pox, ear infections, tonsillitis, upper respiratory tract infections, hormonal disturbances and allergies.¹⁶ Perinatal factors, such as difficult or prolonged delivery, caesarean section delivery, preterm birth and hypocalcemia during the last trimester of pregnancy have been implicated.^{17,18} Postnatal factors may include seizures, antibiotic use during early childhood, various neonatal period problems such as reduced serum calcium levels in a newborn due to delayed response of hormones responsible for its regulation and repeated episodes of high fever usually due to common cold.^{17,18} These factors cause alteration in the sensitive ameloblasts, particularly during the early maturation phase.²

MIH presents as a challenge to the clinicians as the post eruptive enamel breakdown occurs shortly after the eruption of the hypomineralized tooth leading to the hypersensitivity, rapid caries progression and the difficulty in achieving anaesthesia with increase in number of appointments required.^{5,19} Hypomineralization in the second deciduous molar is a significant risk factor for the development of dental caries in the primary dentition in comparison to other deciduous teeth with no defects.²⁰ The lower levels of calcium deposits in the hypomineralized enamel may be responsible for the increased caries occurrence in the affected teeth.²¹

The well established criteria recommended by the EAPD in 2003 for the diagnosis of MIH was used in this report: absence or presence of demarcated opacities, posteruptive enamel breakdown, atypical restoration, extraction due to MIH, failure of eruption of a molar or incisor.¹⁰

Additionally, the hypomineralised areas in this report are classified as severe and moderate.¹¹ All permanent first molars were scored as severe which was attributed to the age of the patient. The child being 8 years of age with the permanent first molars erupted 2 years back may also have resulted in post eruptive enamel breakdown or atypical caries.

There is increased likelihood to develop MIH in children with the hypomineralized second deciduous molars. Children with MIH and DMH usually manifests behaviour management problems and dental fear because of the increased number of the dental visit with the rise in demand for more careful examination at the age of 6-8 yrs, which is the period of the eruption of first permanent molars and incisors.

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