

THE PREVALENCE OF MOLAR INCISOR HYPOMINERALIZATION (MIH) IN A GROUP OF CHILDREN COMING TO THE DEPARTMENT OF DENTISTRY, JAWAHARLAL NEHRU MEDICAL COLLEGE & HOSPITAL, BHAGALPUR, BIHAR

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ABSTRACT:

Objective: The aim of this study was to determine the prevalence of Molar-Incisor Hypomineralisation (MIH) in the Bhagalpur District of Bihar.

Design: Cross-sectional survey of developmental defects of enamel in a random sample of children coming to JLNMC&H during October 2016 to mid – April 2017 were included in the study.

Method: Study information and consent forms were given to every 7 to 15 year old children. Using the modified Developmental Defects of Enamel index, a single dentist examined children in the OPD.

Results: Examination were conducted on 1044 children. MIH prevalence was 14.9%. The prevalence of hypomineralization of any tooth was 15.3%, and that of hypoplasia was 4.0%.

Conclusions: Approximately one in seven children have MIH.

Keywords: Molar-Incisor Hypomineralisation (MIH), modified Developmental Defects of Enamel index hypoplasia



INTRODUCTION:

The term “molar incisor hypomineralization” (MIH) was first used by Weerheijm et al.

in 2001^[1] It denotes enamel defects in first permanent molars and permanent incisors. Molar incisor hypomineralisation (MIH) is the term used to describe a specific pattern of enamel defects. This pattern consists of asymmetric, well-demarcated defects affecting the enamel of the first permanent molars and is

associated with similar defects in permanent incisors and canines.^[2] These defects are seen either as discolored opacities (which are soft and can fracture under masticatory forces) or as a total absence of enamel often involving a significant portion of the enamel crown^[3]

Enamel is derived from ectoderm and produced by ameloblasts. Enamel formation can be divided into the three stages of matrix formation, initial calcification and final maturation though these processes are relatively well

understood, the aetiology of developmental dental defects remains ambiguous. The ameloblasts are highly susceptible to both genetic and environmental influences such as respiratory distress, brain hypoxia and childhood illnesses particularly those associated with fevers, malnutrition or calcium deficiencies. If the disruption occurs during the secretory phase of matrix formation, the teeth are characterized by a deficiency of tooth substance that can range from minor pits and grooves to the total absence of enamel. This type of enamel defect is termed "Enamel Hypoplasia" (Suckling, 1989).^[4] However, if disruption of ameloblasts occurs during either the calcification or maturation phase, the teeth will appear mottled and the enamel will have a qualitative defect (Suckling, 1984b) termed "Enamel Hypomineralisation". Clinically and histologically, combinations of hypoplasia and hypomineralisation may co-exist. More recently, the term "Molar-Incisor Hypomineralisation" (MIH) has been used; this is defined as hypomineralisation and/or hypoplasia of systemic origin of one to four first permanent molars, frequently associated with affected permanent incisors.^[5]

Recent international studies report the prevalence of hypomineralisation of molar FPM with or without the involvement of the incisors in children aged 7 to 13 to be between 4% and 25%. However, only a limited number of studies concerning prevalence data of MIH have been available. Comparable

and representative prevalence studies are lacking.^[4] There is no information about the prevalence of MIH in Bhagalpur, although existing studies of enamel defects provide useful background data.

MATERIALS AND METHODS:

The study was conducted in the OPD of J.L.N.M.C. &H., Bhagalpur, (Bihar) India. All 7 to 15 year-old children coming for the treatment were selected for the study. Prior to the clinical examinations, study information and consent was taken from children's parents/caregivers. Children were considered as having MIH when one or more FPM with or without the involvement of the incisors, when at least one of the incisors is erupted, meeting the diagnostic criteria. All hypomineralised lesions regardless of their size considered for the diagnosis of the defect.

The dental examinations took place at the OPD and were performed in daytime lighting conditions by a single dentist using sterilized mouth mirrors, probe and tweezers. The children were examined with a portable 6V LED 'white' light and an oral mirror while sitting upright in an ordinary school chair. Teeth were cleaned or dried prior to the inspection using gauze. The criteria for classification were based on the Modified Developmental Defects of Enamel Index (mDDE) (Clarkson and O'Mullane, 1989).^[6]

All visible tooth surfaces were examined and any visible developmental defects of

enamel greater or equal to 2 mm in diameter were recorded. Each molar was classified as either unaffected, hypomineralised and/or hypoplastic, or unerupted. The presence of molar tooth restorations was also noted. In cases where the defects were extensive, photographs were taken.

RESULTS:

In total, 1044 (approximately 62.1 % of eligible children) participated in the clinical study. Developmental defects of enamel were observed in 15.9% of participants some 15.3% had hypomineralisation of one or more teeth, and 4.0% had one or more hypoplastic teeth. More females (17.4%) than males (14.2%) had developmental defects of enamel, although this difference was not statistically significant. Hypomineralisation and hypoplasia were most frequently seen in a single tooth. Of the 160 participants with hypomineralization. 47.5% had only one affected tooth; of those with hypoplasia, 61.9% had it in a single tooth. Enamel defects were considerably more common in first permanent molars (14.9%) than in the permanent anterior teeth (3.8%). Some 150 participants (14.4%) had hypomineralisation of the first permanent molars; of these, 30 (2.9%) also had one or more hypomineralised incisors. Of those with hypomineralised incisors, 80.0% also had hypomineralization of the first permanent molar. The association between hypomineralization in at least one incisor and at least one first permanent molar was highly significant. Lower first permanent molars were more frequently affected by MIH (7.5% and 6.9% respectively for teeth 36 and 46)

than upper molar teeth (6.5% for 16 and 5.8% for 26), but this difference was not statistically significant. The anterior tooth most frequently affected by MIH was tooth 21 (1.5% of participants).

	No. of affected	total sample
Any developmental defect of enamel	166	15.9
Unaffected	878	84.1
MIH	156	14.9
Hypomineralization (total no of affected teeth = 326)	160	15.3
First permanent molar	150	14.4
Incisor	38	3.6
Any single tooth	76	7.3
Two teeth	34	3.3
Three teeth	24	2.3
Four or more teeth	26	2.5
Hypoplasia (total no of affected teeth = 72)	42	4.0
First permanent molar	40	3.8
Incisor	4	0.4
Any single tooth	26	2.5
Two teeth	10	1.0
Three teeth	0	0
Four or more teeth	6	0.6

Chart 1: Showing prevalence of molar incisor hypomineralization

DISCUSSION:

The aim of this study was to determine the prevalence of MIH in children of Bhagalpur region and help to establish the extent of the problem. The findings indicate that MIH is common among children of Bhagalpur region, with one in seven having at least one first permanent molar affected. The majority

of international studies of MIH have been conducted on children of European descent (Weerheijm et al. 2003)¹. The prevalence of developmental defects of enamel for the entire sample population was high, at 15.9%. This estimate is comparable to that from a recent study of 9-year-old children in Auckland (NZ) where 19.8% had demarcated opacities. Hypoplasia was found in 4% of children, which is less than the 7.1% reported for the Auckland sample. Schlüter et al also used the mDDE. In 1984, using the DDE index and identical examination methods to this study. Suckling and Pearce investigated developmental defects of enamel in a group of children in the small rural town of Richmond (Nelson, NZ). Direct comparison is difficult because of differences in the definitions used for defects and differences in fluoridation levels. However, they found hypoplasia in 10.7%, and "single demarcated yellow opacities" in 16.5% of participants. Suckling and Pearee, Schlüter et al and Mackay and Thomson all focused on the effect of fluoridation on enamel defects⁸.

Enamel hypoplasia or hypomineralization associated with MIH as shown in this paper can appear on a single tooth or a number of teeth in the dental arch and is as commonly observed in optimally fluoridated areas as in low-fluoride areas (Clarkson and O'Mullane 1989)⁶. Our findings on MIH in Bhagalpur region are comparable to those of international studies of MIH prevalence in children aged 6-12. Despite cultural and

environmental differences and variations in classification criteria among these countries and studies, the various MIH prevalence estimates generally fall within about 6% of each other, which is remarkably consistent. There was a negligible (0.4%) difference in defect frequency between the upper and lower jaws. This concurs with the findings of Wehrcijm et al (2001a) and Jalevik et al (2001) but differs from those of Leppaniemi et al (2001). Schulte et al (2008) and Suckling et al (1984)⁴, who reported defects in maxillary molars to be approximately 4% more frequent than for mandibular molars. The enamel formation of maxillary and mandibular teeth occurs at different times (Schour and Massier. 1940). The between-jaw distribution of teeth with developmental defects of enamel may give an indication of the time at which an external etiological factor occurred. Alternatively, any difference in prevalence between the jaws could simply reflect errors in measurement and sampling.

CONCLUSION:

The findings of this study indicate that hypomineralization and hypoplasia are moderately prevalent in children of Jammu region. The presence of either appears to be associated with a greater susceptibility to permanent dentition caries which will, in turn, result in greater ongoing need for dental care in affected children. Based on our findings and other studies over the last decade, one in seven children (on average) have MIH).

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