

Prevalence of Hypomineralised Second Primary Molar and its Possible Association with Molar Incisor Hypomineralisation in Children Aged 6-9 Years of Mehsana District, Gujarat, India: A Cross-sectional Study

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ABSTRACT

Introduction: Dental enamel in primary teeth may exhibit a developmental defect known as Hypomineralised Second Primary Molars (HSPM), which presents with clinical signs and symptoms similar to Molar Incisor Hypomineralisation (MIH). Second Primary Molars (SPMs) may serve as a reliable indicator for predicting future hypomineralisation of permanent incisors and molars prior to their eruption. Early identification of HSPM can aid in anticipating future dental challenges such as tooth sensitivity, increased susceptibility to caries and restorative complications in the permanent dentition. Currently, there is limited literature regarding the prevalence of HSPM in Gujarat, India.

Aim: To determine the prevalence of HSPM and its possible association with MIH among children 6-9 years in the Mehsana District of Gujarat, India.

Materials and Methods: A cross-sectional study was conducted among children aged 6-9 years in the Department of Paediatric and Preventive Dentistry, Narsinhbhai Patel Dental College and Hospital, Sankalchand Patel University, Visnagar, Gujarat, India. The district was divided into three zones and a total sample size of 840 children was calculated. Each zone included 280 children selected from both private and government schools. The

European Academy of Paediatric Dentistry (EAPD) guidelines were followed for the assessment of both MIH and HSPM, wherein only First Permanent Molars (FPMs) and SPMs were assessed. The collected data were tabulated and analysed using the Chi-square test and the Odds Ratio (OR) was calculated to assess the association between MIH and HSPM. A p-value of <0.05 was considered statistically significant.

Results: The study revealed a prevalence of 36/840 (4.2%) for HSPM and 44/840 (5.2%) for MIH in the Mehsana District. Both MIH and HSPM were present in 22/54 (40.7%) of the affected children, suggesting a strong association between the two conditions (OR=44.75; p<0.001; 95% CI=22.55-105.38). The most common type of defect observed in SPMs was white creamy opacities (47.36%), followed by yellowish-brown lesions (31.57%).

Conclusion: The present study highlights a notable prevalence of HSPM and MIH among children aged 6-9 years in the Mehsana district, with a significant association between the two conditions. Early identification of HSPM may serve as a predictive marker for MIH, emphasising the importance of timely diagnosis and preventive care to mitigate future dental complications in the permanent dentition.

Keywords: Developmental defects of enamel, Enamel hypomineralisation, Primary dentition, Tooth demineralisation

INTRODUCTION

In recent years, several terms—such as non fluoride enamel opacities, opaque spots, cheese molars, internal enamel hypoplasia, non endemic enamel mottling and idiopathic enamel opacities—have been used to describe non fluoride-associated developmental defects of tooth enamel. Currently, MIH is the preferred term for a major subset of these enamel developmental disturbances [1].

Disruption during the initial calcification and/or maturation phases of enamel formation can result in a qualitative defect known as enamel hypomineralisation. When this condition affects FPMs and incisors, it is termed MIH. The SPMs, affected by a similar defect, are referred to as HSPM [2].

Because the mineralisation of FPM crowns and SPMs occurs simultaneously, disturbances during this critical developmental window—particularly during prenatal and neonatal periods—can adversely affect the mineralisation of both dentitions [3].

Enamel lesions in HSPMs include well-demarcated opacities that are creamy white or yellow-brown, atypical restorations and lesions associated with Post-Eruptive Breakdown (PEB). These features

closely resemble those observed in MIH. HSPMs are therefore more susceptible to PEB and have an increased risk of dental caries [4]. Globally, the prevalence of HSPM varies widely, reflecting variations in study design, population characteristics, ethnicity, age groups and diagnostic criteria. A recent systematic review and meta-analysis of 37 studies involving more than 26,000 children reported a pooled global prevalence of HSPM of 4.08% at the tooth level and 6.8% at the child level [2].

Studies from Aydin and the Netherlands reported lower prevalence rates of HSPM, with 4.4% among schoolchildren in Aydin [5] and 4.9% among five-year-old children in the Netherlands [6]. Lower-to-middle prevalence rates (5-10%) have been reported as 5.0% among children aged 6-10 years in India (Delhi NCR) [7] and 6.6% among Iraqi schoolchildren in Baghdad [8]. Similarly, prevalence rates among Saudi Arabian children ranged from 5.4% [9] to 11.4% [10].

Literature reviews have reported HSPM prevalence rates ranging from 2.7% to 21.8% across different global populations [11]. South American studies report significantly higher prevalence, including 20% among children aged 6-12 years in Venezuela [12] and

24.5% among preschool children in Brazil [13]. Exceptionally high rates have been observed in Syrian preschoolers in Aleppo, with a reported prevalence of 41% [14].

These variations highlight the significant geographic differences in HSPM prevalence and emphasise the need for more robust population-based studies and standardised diagnostic criteria to accurately estimate the global burden of the condition.

The aetiology of MIH is multifactorial, with clinical variability influenced by the timing, duration and severity of aetiological factors. Potential contributing factors include prenatal and perinatal conditions such as low birth weight, preterm birth, caesarean delivery, maternal sickness and perinatal hypoxia. Additionally, childhood illnesses occurring during the critical developmental window for MIH—from birth to four years of age—such as measles, bronchitis, otitis media, pneumonia, asthma, urinary tract infections, fever, renal disorders, gastrointestinal disturbances and antibiotic use, have been implicated. Recent research has also highlighted the role of genetic susceptibility and epigenetic influences in the development of MIH [10].

Early identification of affected children and appropriate management can make the condition easier to treat and help prevent possible adverse outcomes and the associated high healthcare costs [15]. The signs and symptoms of HSPM are similar to those of MIH and include hypersensitivity, increased susceptibility to dental caries, restorative challenges and behavioural issues in children [6]. There is a lack of documented data regarding the prevalence of HSPM in Gujarat. Therefore, the present study aimed to assess the prevalence of HSPM and MIH and to evaluate their possible association in the Mehsana District of Gujarat.

MATERIALS AND METHODS

The present cross-sectional study was conducted in the Department of Paediatric and Preventive Dentistry, Narsinhbai Patel Dental College and Hospital, Sankalchand Patel University, Visnagar, Gujarat, India. The district comprises 10 talukas, which were grouped into three zones: North (4 talukas), Central (2 talukas) and South (4 talukas). One taluka from each zone was randomly selected—Unjha, Visnagar and Kadi, respectively. According to the Census of India (2011), the population of the district is approximately 2.035 million, with groundwater fluoride levels ranging from 1.5 to 5.6 mg/L [16,17]. The study was conducted from June 2024 to October 2024 after obtaining ethical clearance from the Institutional Ethics Committee (IEC) of Narsinhbhai Patel Dental College and Hospital, Visnagar, Gujarat, India (Approval No. NPDC/IEC/2023/117). Written informed consent was obtained from both school authorities and parents prior to conducting oral examinations.

Sample size calculation: The sample size was calculated using the formula:

$$n=Z^2p(1-p)/d^2$$

Assuming a prevalence of 30% (p=0.30), based on previously reported prevalence ranges (4-41%) [5,14], with a 95% confidence level (Z=1.96) and a precision of 3.11% (d=0.0311), the required sample size was approximately 835. Considering feasibility at the ground level, a final sample size of 840 children was included.

Inclusion criteria: School-going children aged 6-9 years with no previous restorations on the reference teeth were included. Children were required to have at least one erupted FPM. Additionally, children who had been residents of the study area since birth and whose parents had resided in the region for at least 10 years were included. This information was obtained through telephonic interviews using contact details provided by the school authorities.

Exclusion criteria: Children with hypoplastic lesions on reference teeth, tetracycline staining, or other developmental dental abnormalities were excluded. Children whose FPMs and/or SPMs were severely compromised, missing, or whose reasons for tooth loss were unknown were also excluded from the study.

Study Procedure

Study population: A total of 1,460 children were initially screened, of whom 840 met the inclusion criteria and were enrolled in the study. The sample was evenly distributed across the three zones—North, Central and South—with 280 children selected from each zone. To ensure uniform distribution, every sixth child within the target age group was included.

To ensure adequate geographic and demographic representation, four schools from each zone were randomly selected based on the total number of schools in each region.

Although the calculated sample size was achieved, several feasibility challenges were encountered during data collection. These included absenteeism of children on examination days, lack of parental consent in some cases and exclusion of children who did not meet the eligibility criteria. Additional logistical constraints, such as limited examination time, coordination with school authorities and restricted access to certain schools, also influenced the screening process.

Calibration: All examinations were conducted by a single calibrated examiner who had received comprehensive training based on the European Academy of Paediatric Dentistry (EAPD) guidelines [18]. The use of a single examiner was intended to minimise inter-examiner variability and ensure consistency in the diagnosis and scoring of MIH and HSPM, thereby enhancing the internal validity of the study.

Intra-examiner reliability was assessed by re-examining 15% of the total sample on the final examination day at each institution. According to the criteria proposed by Landis JR and Koch GG (1977), the intra-examiner kappa value was 0.86, indicating “almost perfect” agreement [19].

Study setting and diagnostic criteria: Oral examinations were conducted within the school premises, with children seated on upright chairs. The examinations followed World Health Organisation (WHO) criteria and were performed using a blunt probe, a dental mirror and artificial illumination on clean teeth under moist conditions.

Hypomineralisation defects of both FPMs and SPMs were recorded using the modified EAPD diagnostic criteria [Table/Fig-1] for MIH in FPMs [20]. Teeth exhibiting PEB were considered more severely affected than those without breakdown and yellowish-brown opacities were classified as more severe than creamy white opacities.

Each tooth should be examined for:
1. Absence or presence of demarcated opacities (defect altering the translucency of the enamel)
2. Posteruptive enamel breakdown (loss of surface enamel after tooth eruption, usually associated with a pre-existing opacity)
3. Atypical restorations (frequently extended to the buccal or palatal smooth surfaces reflecting the distribution of hypoplastic enamel)
4. Extracted molars due to MIH.
5. Failure of eruption of a molar or incisor
Permanent first molars and incisors (12 index teeth) should be examined and therefore the age of 8 years upwards is the best time for examination.
Examination for MIH should be performed on wet teeth after cleaning and clearly visible opacities regardless of size should be recorded.

[Table/Fig-1]: Codes and clinical status assessment criteria for MIH.

To maintain sterilisation protocols, all instruments were disinfected using Dettol solution diluted with water. Immediate care and referral were provided to children when required.

STATISTICAL ANALYSIS

The complete dataset was initially recorded on a structured, pre-printed proforma that included sections for documenting demographic details of each participant, as well as the type, severity and location of tooth or surface defects. The collected data were subsequently entered into a Microsoft Excel spreadsheet. The

geographic distribution and prevalence of HSPM were statistically analysed. The Chi-square test was applied and p-values less than 0.05 were considered statistically significant. The association between HSPM and MIH was evaluated using the OR with a 95% Confidence Interval (CI) using Statistical Package for the Social Sciences (SPSS) software version 20.0.

RESULTS

Demographic data: The demographic characteristics of the study participants is presented in [Table/Fig-2]. A total of 840 children aged 6-9 years were included in the study. The mean age of the participants was 7.80±0.810 years.

Age (years)	Male (n)	Female (n)	Total (N)
6	40	40	80
7	110	110	220
8	140	140	280
9	130	130	260
Total	420	420	840

[Table/Fig-2]: Demographic data of subjects included in the study.

Prevalence: Well-defined hypomineralised enamel defects were identified in 54 children. As shown in [Table/Fig-3], of these 54 participants, 44 were affected by MIH and 36 by HSPM. The overall prevalence in the analysed population was 5.2% for MIH and 4.2% for HSPM. Concurrent presence of both MIH and HSPM was observed in 22 children (40.7%).

Subjects	n (%)
Total no. of subjects affected	54 (6.4%)
Total no. of subjects affected by HSPM	36 (4.2%)
Total no. of subjects affected by MIH	44 (5.2%)
Total no. of subjects affected by both	22 (2.61%)

[Table/Fig-3]: Prevalence of MIH and HSPM at subject level N=840.

The prevalence of HSPM and MIH at the tooth level. MIH affected 132 of 144 teeth, while HSPM affected 114 of 144 teeth is illustrated in [Table/Fig-4].

Group	Subjects affected n (%)	Teeth affected n (%)	Mean teeth per affected subject
HSPM only	36 (4.2%)	114/144	3.17
MIH only	44 (5.2%)	132 /144	3.00

[Table/Fig-4]: Prevalence of MIH and HSPM at tooth level.

Defects observed in HSPM: As shown in [Table/Fig-5], 13 boys (36.11%) and 23 girls (63.89%) were affected by HSPM. The prevalence was significantly higher in girls than in boys (p=0.0071).

Subjects affected	n (%)	p-value
Males	13 (36.11%)	0.0071*
Females	23 (63.89%)	

[Table/Fig-5]: Subjects affected by HSPM only N=36.

*Significant p-value, calculated using Chi-square test

Out of the total 144 SPMs examined in 36 children (four SPMs per child), 114 teeth (79.16%) were affected by HSPM. Among these, 51 were maxillary SPMs and 63 were mandibular SPMs [Table/Fig-6]. Although mandibular SPMs were more frequently affected than maxillary SPMs, the difference was not statistically significant.

The most prevalent characteristic defects, assessed using the EAPD criteria [20], in descending order of frequency, were creamy white opacities 54 (47.36%), yellowish-brown opacities 36 (31.57%), creamy white opacities with Post-Eruptive Breakdown (PEB) 13 (11.40%), yellowish-brown opacities with PEB 9 (7.89%) and atypical restorations 2 (1.75%), as depicted in [Table/Fig-7].

Teeth	n (%)	p-value
Maxillary	51 (44.7)	0.055
Mandibular	63 (55.2)	

[Table/Fig-6]: Number of teeth affected by HSPM (n=114/144). Calculated using Chi-square test

Defects	n (%)
Creamy white	54 (47.36)
Yellow brown	36 (31.57)
Yellow brown with PEB	9 (7.89)
Creamy white with PEB	13 (11.40)
Atypical restorations	2 (1.75)

[Table/Fig-7]: Distribution of defect characteristics seen in HSPM (N=114).

Subjects affected by both MIH and HSPM: MIH and HSPM were concurrently present in 22/54 (40.7%) of affected participants. The OR for MIH was significantly higher in children with HSPM (OR=44.75; p<0.001; 95% CI=22.55-105.38). The OR was selected to assess the association between MIH and HSPM because of the cross-sectional study design, which allowed comparison of the odds of MIH occurrence in children with and without HSPM. An OR greater than 1 indicated a positive association between MIH and HSPM.

Further analysis based on the number of HSPMs per child revealed that participants with two or more HSPMs had a significantly higher OR for MIH (p<0.001), whereas the presence of a single HSPM was not significantly associated with MIH (p=0.183), as shown in [Table/Fig-8].

Total no. of subjects	No. of subjects with HSPM				
	No HSPM	One HSPM	Two HSPM	Three HSPM	Four HSPM
No MIH	32	6	4	3	3
MIH	22	1	9	5	7
OR		5.91	79.77	59.09	82.73
95% CI		0.68 – 51.19	22.82 – 278.91	13.28 – 262.92	20.05 – 341.35
p-value		0.183	<0.0001*	<0.0001*	<0.0001*

[Table/Fig-8]: Distribution of HSPM in subjects with or without MIH.

*Significant p value, OR – Odd's ratio, CI- Confidence Interval

Overall OR and CI for the lesion was calculate and the values were- OR 44.75; p<0.001; 95% CI = 22.55-105.38

DISCUSSION

Research on the prevalence and characteristics of non fluoride-related developmental enamel defects has gained considerable international attention following the introduction of EAPD assessment criteria. Ghanim A et al., first introduced the term MIH in 2001 to describe sporadic and significant enamel defects affecting one or more FPMs and/or permanent incisors [1].

The term HSPM was later introduced to describe MIH-like demarcated opacities affecting SPMs. This similarity may be attributed to the overlapping periods of mineralisation during tooth development [6].

The present study involved children aged 6-9 years from various schools in the Mehsana District and reported a prevalence of 4.2% for HSPM. This finding is comparable to studies by Estivals J et al., (2008), which reported a prevalence of 9.5% and Gambetta-Tessini K et al., (2018), which reported an 8% prevalence of HSPM among children aged 6-12 years [21,22].

Similarly, the overall prevalence of MIH in the present study was 5.2%, which falls within the reported range of 2.4-40.2% documented by Subramaniam P et al., (2016) [23]. The prevalence observed in this study is also consistent with findings reported by Allazzam SM et al., (2014), who reported an 8.6% prevalence among children aged 8-12 years and Ahmadi R et al., (2012), who observed MIH in 12.7% of Iranian children aged 7-9 years [24,25].

The variability in prevalence rates reported across studies may be attributed to several factors, including differences in diagnostic criteria [19], examiner calibration and bias [1], population and geographic variability [26] and variations in sample size and study design [27].

The study was conducted among children aged 6-9 years, as MIH and HSPM can be most accurately diagnosed when the FPMs are fully erupted, typically around eight years of age. If examinations are performed at a later stage, PEB may occur, potentially masking hypomineralisation and leading to underestimation of prevalence [28].

In the present study population, a total of 36 participants were identified with HSPM. Females (63.89%) were significantly more affected than males (36.11%). Maxillary SPMs (44.7%) were less frequently affected than mandibular SPMs (55.2%). These findings are consistent with studies conducted in Kaunas by Jasulaityte L et al., (2007) and in Udaipur by Bhaskar SA and Hegde S (2014) [29,30]. However, the difference between maxillary and mandibular arches was not statistically significant ($p>0.05$).

The exact reason for gender differences in HSPM prevalence remains unclear. However, according to Ortega-Luengo S et al., the higher prevalence observed in girls may be attributed to earlier dental maturation, resulting in longer post-eruption exposure and increased susceptibility to enamel loss [31].

The severity of both HSPM and MIH ranges from demarcated opacities to structural enamel loss [1]. In the present study, the most commonly observed defects in HSPM were creamy white opacities 54 (47.36%), followed by yellowish-brown opacities 36 (31.57%), creamy white opacities with PEB 13 (11.40%) and atypical restorations 2 (1.75%).

The findings revealed an overall trend indicating that the number of molars affected by HSPM was directly correlated with defect severity, consistent with studies by Estivals J et al. and Ben Salem J et al., (2023) [21,32]. Children with one or two affected molars were less likely to exhibit PEB, whereas those with more than three affected molars tended to present with mild to severe forms of HSPM.

Several previous studies have investigated the association between MIH and HSPM, including those by Ghanim A et al., in 2015, Elfrink MEC et al., in 2012 and Mittal N and Sharma BB in 2015 [1,6,33]. The present study demonstrates a significant association between HSPM and MIH, aligning with the findings of Elfrink MEC et al. and Mittal N and Sharma BB [6,33]. Differences between the present study and the findings of Ghanim A et al., may be attributed to variations in age groups studied and the higher prevalence of MIH reported in their research [1].

In the current study, the presence of HSPM was associated with an OR of 44.75 for the occurrence of MIH. The OR was used because it quantifies the strength of association between an exposure (presence of HSPM) and an outcome (development of MIH). ORs are commonly employed in epidemiological studies to determine how much more likely an outcome is in an exposed group compared to a non exposed group [34].

Further analysis demonstrated that participants with two or more HSPMs had significantly higher odds of developing MIH ($p<0.001$), indicating a strong association between increasing numbers of affected primary molars and MIH. Conversely, the presence of a single HSPM was not significantly associated with MIH ($p=0.183$), suggesting that isolated HSPM may not be a reliable predictor of MIH.

An important consideration in interpreting enamel hypomineralisation data is fluoride exposure, particularly from groundwater sources. The present study was conducted in a region with variable groundwater fluoride concentrations ranging from 1.5 to 5.6 mg/L. Fernandes IC et al., (2021) reported that fluoride concentration in drinking water did not significantly influence MIH prevalence in Brazil; however, children with dental fluorosis exhibited more severe MIH, suggesting a potential modifying effect of fluoride on defect severity [35].

Nevertheless, further research is warranted to better understand the influence of HSPM on primary dentition and its long-term implications for dental health. Additional studies involving diverse age groups and populations with varying caries experiences would contribute to a more comprehensive understanding of the condition and its clinical management.

Clinical significance:

- The HSPM frequently occurs in children with a high caries risk. The present study, consistent with existing literature, demonstrates a significant association between HSPM and MIH, facilitating early identification of children at risk for MIH.
- The HSPM is often misdiagnosed as early childhood caries. Therefore, paediatric dentists should be well-versed in recognising the clinical features of HSPM to implement appropriate preventive strategies for high-risk children.
- Children with HSPM are more likely to develop MIH in the future; hence, clinicians should closely monitor these patients during the eruption of FPM.

Limitation(s)

The cross-sectional design of the study limits causal inference, allowing only the identification of associations rather than cause-and-effect relationships. The sample was restricted to a single district (Mehsana), which may limit generalisability to other populations or geographic regions. Environmental confounding factors such as nutrition, systemic illnesses and prenatal or postnatal influences were not comprehensively assessed and may have affected enamel development. Although groundwater fluoride levels were reported, individual fluoride exposure through diet, toothpaste, or supplements was not measured, potentially confounding the results. Additionally, the study did not evaluate specific tooth surfaces affected, limiting insights into the distribution and severity of enamel defects.

CONCLUSION(S)

The findings of the present study support the role of HSPM as a clinical predictor of MIH. Children exhibiting HSPM demonstrated a significantly higher likelihood of developing MIH, emphasising the importance of early identification and monitoring for timely preventive and therapeutic interventions. These results reinforce existing evidence and suggest that incorporating HSPM assessment into routine paediatric dental examinations may enhance preventive strategies and improve long-term oral health outcomes.

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