Original Article

Molar Incisor Hypomineralisation (MIH): A Review

Dr. Amitava Bora[1*], Dr. Piyali Datta[2], Dr. Rajib Saha[3], Dr. Kaushik Dutta[4]

[1*]Assistant Professor, Department. of Pediatric and Preventive Dentistry, Burdwan Dental College and Hospital.
Email: dramitavabora@gmail.com
[2]Reader, Dept. of Pediatric and Preventive Dentistry, Guru Nanak Institute of Dental Sciences & Research, Kol-700114
Email: dr.piyalidatta.datta@gmail.com
[3]Reader, Dept. of Pediatric and Preventive Dentistry, Guru Nanak Institute of Dental Sciences & Research, Kol-700114
Email: rajib.saha123@yahoo.in
[4]Professor and HOD, Dept. of Oral Medicine and Radiology, Guru Nanak Institute of Dental Sciences & Research, Kol-700114

***Corresponding Author:** Dr Amitava Bora

*108/6 Nagendra Nath Road, Kolkata – 700028, West Bengal, India. Contact no- 9432109147, 03325002620,
Email id: dramitavabora@gmail.com
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Abstract

Molar incisor hypomineralisation (MIH) is a highly prevalent developmental defect of one or upto four first permanent molars and permanent incisors. Its clinical implications are hypersensitive teeth, rapid progression of caries, impairment of mastication due to rapid attrition and aesthetic impairment which lead to complexity in treatment planning, poor prognosis of the restorations, difficulty in achieving pain control during treatment and behaviour management problem. Clinically the defect presents as opaque lesion varying in colour from white to yellow or brown and with a sharp demarcation between the affected and sound enamel to post eruptive enamel breakdown. The main objective of this paper is to review the etiopathogenesis, prevalence, clinical features, diagnostic features and treatment approach.

Key word: Molar- incisor- hypomineralisation, enamel hypoplasia, Post eruptive enamel breakdown

INTRODUCTION:

Molar incisor hypomineralisation (MIH) is a distinct clinical entity defined by hypomineralisation of systemic origin affecting one, two or all first permanent molars and the permanent incisors[1]. Hypomineralised enamel defects can range from mild opacities, whitish or yellowish discoloration, to severe enamel involvement, which break down rapidly after eruption[2]. For a patient to be diagnosed as suffering from MIH, they have to have at least one first permanent molar affected with or without the involvement of the incisors[2]. However, if a patient has opacity affecting the incisors only, the condition is not MIH. Patient with MIH presents several clinical problems, including rapid wear, enamel loss, increased susceptibility to caries and sensitivity. Patients frequently claim esthetic problem when anterior teeth are involved[3]. A variety of systematic medical factors have been proposed as contributing factors for MIH, including prenatal, perinatal and post natal illnesses, low birth weight, antibiotic consumption and toxins from brestfeeding[4].
Today MIH is considered to be a very important clinical entity as they cause tooth defect ranging from mild opacities to severe irreversible enamel wear and diagnosis of this condition is very challenging. Moreover there is no fixed universally accepted treatment guideline. A proper knowledge regarding prevalence and etiological factors are very important as that would help in diagnosing and deciding the treatment plan. The relatively high prevalence of MIH around the globe and ignorance among general population as well as medical professionals make this clinical condition more important.

METHODOLOGY USED IN THE REVIEW PROCESS:

A broad search of Pubmed, Pubmed central, MedLine, Research Gate, EBSCOhost, Google Scholar, Web of Science Data bases was conducted for the years 1980 until 2022, using as index terms 'Developmental enamel defect' , ' Molar Incisor Hypomineralisation', 'Non fluoride hypomineralisation of permanent teeth', 'Idiopathic hypomineralisation of permanent teeth', 'Hypomineralised permanent molars' and 'Hypomineralised permanent incisors', 'MID', 'Prevalence, Etiology, Management of Molar- Incisor- hypomineralisation'. Papers other than in english were excluded.

DEFINITIONS:

Molar incisor hypomineralisation (MIH) was first recognised in the late 1970s by Swedish dentists working the public dental services. Due to the lack of an agreed definition, prior to 2001, the literature regarding MIH is confusing and it is difficult to be sure wheather different researchers were referring to the same thing. Several terminologies were used for describing this condition by different researchers like non fluoride enamel opacities, internal enamel hypoplasia, nonendemic moltting of enamel, opaque spots, idiopathic enamel opacities, enamel opacities or cheese molars[5]. The term molar incisor hypomineralisation was first cited by Weerheijm et al[1]
(2001),

Molar incisor hypomineralisation (MIH) is defined as the developmentally derived dental defect that involves hypomineralisation of 1 to 4 permanent first molars, frequently associated with similarly affected permanent incisors[6,7.] (Weerheijm et al.2003).

PREVALENCE (Table 1 and Table 2):

MIH is recognized as a global dental problem and epidemiologic reports from all over the world are continuously publishing (Jalevik 2010). Prevalence rates ranges from 2.5-40.2% using initial presentation and establishment of diagnostic criteria for MIH[8].
Prevalent rates of various studies around the globe and in India is given in table 1 and table 2.

ETIOLOGY AND PATHOGENESIS:

Etiological factors of causing MIH are still not very clear. It is hypothised that MIH might have a multifactorial etilogy acting additionally or even synergistically with a genetic predisposition[50,51].
In general, systemic factors that disturb the ameloblasts during the secretory phase cause restriction of crystal elongation and resulting pathologically thin or hypoplastic enamel. On the other hand disturbances during the calcification and/ or maturation stage of amelogenesis resulting pathologically soft (hypomatured, hypomineralised) enamel of normal thicknesses. According to Reid and Dean[52], enamel formation as a whole takes approximately one thousand days (from 18 day IU to 3 years of life) in permanent 1st molar and incisors. Generally factors acting within this time period lead to enamel defect in permanent 1st molar and incisors. Hypomineralisation may also develop later because enamel maturation in the first permanent molars takes several years[53].

Factors causing MIH

At Prenatal period

The last trimester of pregnancy is a critical period during which amelogenesis of first permanent molars and incisors starts. It was suggested that maternal disorders during pregnancy such as Cardiorespiratory diseases, maternal hypertension, infections of the urinary tract, A and D vitamins deficiency, anaemia, toxicity, diabetes mellitus, rubella embrayopathy, chicken pox, maternal high fever during pregnancy, chronic use of myometrium spasmolytic medication might result in developmental defects in the child[53,54].

At Perinatal period

During this term, various medical conditions Caesarean section, prolonged delivery, complicated delivery, preterm delivery and twin deliveries etc may act as causative factors[51,53].
Preterm or complicated delivery may lead to conditions like prolonged respiratory suppression and hypoxia inhibiting the action of the proteolytic enzymes and hampering development of crystal hydroxyapatite resulting to enamel hypomineralisation[54]. In a study it was shown that compared with newborns vaginal delivery those delivered by elective caesarean section had an increased risk of overall and serious respiratory illnesses, conditions associated with hypoxia ultimately resulting in enamel defect[54,55].

At Post natal period:

Frequencies of MIH in children with a systemic disease history in the first three years of age is higher than those without a disease history[56].
Prolonged childhood illnesses, hyper pyrexia, repeated / prolonged medications (antibiotic like amoxicillin)[56], fluoride use, breastfeeding and exposure to environmental contaminants such as polychlorinated biphenyls and polychlorinated dibenzop-dioxins/ dibenzofurans (dioxins), infections such as otitis media, pneumonia, asthma, bronchitis, upper respiratory tract infections, urinary tract infections and exthanthomatous diseases like chicken pox, rubella, measles are probable causative factors.[56,57]
Hypocalcaemia, febrile and afebrile seizure, nutritional deficiencies, brain injury and neurology defects, cystic fibrosis, syndromes of epilepsy and dementia, atopia, lead poisoning repaired cleft lip and palate, radiation treatment, epidermolysis bullosa, celiac disease and gastro intestinal disorder have been suggested as other possible causes.[50.51,58]
Defects in genetic structure affecting enamel formation and maturation (Mmp20. Klk4. Dlx gene) also may cause such enamel defects.[59,60]

CLINICAL PRESENTATION

MIH is connected to a number of subjective and objective problems like loss of tooth substance, increased risk of plaque accumulation and dental caries, severe discomfort, hypersensitivity, pain, loss of restorations, esthetic problem and difficulty in achieving pulpal anaesthesia during treatment.
The clinical appearance of MIH starts as demarcated opacity on the affected tooth with a white cream to dark yellow colour, but they always show a sharp demarcation between the affected and sound enamel. The opacities are usually limited to the incisal or cuspal one third of the crown, rarely involving the cervical one third. The tooth surface enamel initially develops to a normal thickness but can chip off under masticatory forces causing post eruption breakdown (PEB). The severity of MIH may vary considerably and one to all four of the permanent first molars may be affected in different individuals[58,61].

Diagnosis of MIH:

Early diagnosis and proper preventive approaches are the most important aspect of management of any diseases. Any examination for MIH should be under taken on clean wet teeth and the age of eight years is optimal, as at this age all permanent first molars and most of the incisors are erupted.[62]

There are several diagnostic criteria are available for diagnosis of MIH like modified defect of dental enamel (MDDE) index given by Federation dentaire International (Table 3)[8] and the criteria of Weerheijm et al.[62] (table 4). But the modified development dental enamel (MDDE) index and criteria Weerheijm et al[62] are considered to be too time consuming and not adequate for MIH prevalence studies because the post eruptive breakdown is a pathognomonic feature in MIH but the MDDE index does not clearly distinguish PEB from enamel hypoplasia. EAPD guideline[63] (Table 5) is more comprehensive in that aspect and followed widely. Nowadays to simplify the use of MIH scores the severity of MIH can be determined by dividing the affected teeth in only two groups: mild defect (demarcated opacities) and moderate/ severe defects (enamel breakdown and atypical restorations)[62,63].

MIH- Molar Incisor hypomineralisation
PEB- Post eruptive enamel breakdown
EAPD- European Academy Of Pediatric Dentistry

MANAGEMENT OF MIH

Treatment modalities are broadly divided into three aspects; prevention, restoration and extraction.

Preventive approach:

Different preventive methods are

1. Dietary counselling to prevent Dental caries or further decay.
2. Fluoride tooth paste are recommended.
3. In cases of dental sensitivity desensitizing toothpaste with remineralising agents like casein phosphopeptide-amorphous calcium phosphate (CPP-ACP) are indicated.[64,65,66]
4. Fissure sealants should be applied early after molars eruption and before enamel breakdown.
5. As suggested by Ligydikis et al., 2010[66], at late mixed dentition, incisors with whitish-creamy opacities may occasionally respond to bleaching with carbamide peroxide for esthetic improvement.
6. Another conservative approach is microabrasion with either 18% hydrochloric acid or 37% phosphoric acid and pumice for 60s. ((Lygidakis et al., 2010[66], Willmott et al., 2008[67]).

Restoration with adhesive materials

Restoration of the affected first permanent molars can be complicated by difficulties in defining the cavity margins and the choice of the suitable replacement material. Concerning the difficulties in defining cavity margins , two approaches have been proposed: removal of all defective enamel until sound surfaces are reached [William et al[68]] and removal of the porous enamel only, until resistance to the bur or to the probe is felt [Fayle, 2003[69;] Lygidakis et al., 2003[70]].

The first approach means that a lot of tooth material is lost but is better if an adhesive material is used for bonding to enamel. The second approach is less invasive, but it can mean that the defective enamel may continue to chip away during loading.

The different adhesive restorative materials for the restorative purpose of MIH are Glass Ionomer Cement, Resin Modified Glass Ionomer Cements (RMGIC), Polyacid modified composite Resins (PMCR), Composite resins (CR). Amalgam is a non-adhesive material and its use in these atypically shaped cavities is not indicated. Fayle, 2003[69]; William et al., 2006[68;] Croll 2000[71]].

GIC has been additionally proposed as an intermediate layer, prior to composite placement, in cases that the cavity involves large areas of dentine [Mathu- Maju and Wright, 2006[72]].

Preformed metal crowns for First Permanent Molars have been used for many years to cover molars with defective enamel and they are still recommended as a treatment option for MIH posterior teeth [William et al., 2006[68;] Fayle, 2003[69];].

In hypomineralsed Incisors conservative approach should be used as the first line of treatment before more invasive treatment such as resin restorations/veneers or crowns[73].

Pulp Therapy:

In case of pulp exposure based on extent different pulp therapies are indicated[74].

Extraction:

Timely extraction is a feasible treatment option in cases of severe hypomioneralization, severe pulpal pain, large multisurface lesions, non restorable tooth, inability to achieve pulpal anaesthsia, apical pathosis, behaviour management problem etc. When extraction has to be done early orthodontic assessment is recomended[53,56]

If the orthodontic condition is favorable, the ideal dental age for extracting the defective non restorable FPM would be 8.5 to 9 years of age[75].

CONCLUSION

The prevalence and effect of MIH is a matter of concern especially for paediatric population. Moreover several etiological factors and various clinical forms makes the diagnosis complicated. So a proper knowledge of prevalence, pathogenesis and clinical manifestation is necessary for early diagnosis and treatment planning.

REFERENCES:

1. Weerheijm, K.L., Jalevik, B., Alaluusua, S. Molar-incisor hypomineralisation. Caries Res 2001; 35 (5): 390-391.
2. Fitzpatrick L, O'Connell A. First permanent molars with molar incisor hypomineralisation. J Ir Dent Assoc. 2007 Spring;53(1):32-7
3. Lygidakis NA, "Treatment modalities in children with teeth affected by molar-incisor enamel hypomineralisation (MIH): a systematic review," European Archives of Paediatric Dentistry, vol. 11, no. 2, pp. 65–74, 2010.
4. William V, Messer LB, Burrow MF: Molar-incisor-hypomineralisation Review and recommendations for clinical management. Paediatr Dent 2006;28(3):224-32.
5. Croll TP. Creating the appearance of white enamel dysmineralization with bonded resins. J Esthet Dent 1991;3:30-3.
6. Weerheijm KL. Molar-Incisor-Hypomineralisation (MIH). Eur J Paediatr Dent 2003;4:114-20.
7. Mathu-Muju K, Wright JT. Diagnosis and treatment of molar incisor hypomineralization. Compend Contin Educ Dent. 2006;27:604–10.
8. Jalevik B. Prevalence and Diagnosis of Molar-Incisor- Hypomineralisation (MIH): A systematic review. Eur Arch Paediatr Dent. 2010 Apr;11(2):59-64.
9. Leppaniemi A, Lukinmaa PL, Alaluusua S. Nonfluoride hypomineralizations in the permanent first molars and their impact on the treatment need. Caries Res 2001 JanFeb;35(1):36-40.
10. Zagdwon AM, Toumba KJ, Curzon ME. The prevalence of developmental enamel defects in permanent molars in a group of English school children. Eur J Paediatr Dent 2002;3:91-6.
11. Preusser SE, et al.; Prevalence and severity of molar incisor hypomineralization in a region of Germany -- a brief communication. J Public Health Dent. 2007
12. Kemoli AM. Prevalence of molar incisor hypomineralisation in six to eight year-olds in two rural divisions in Kenya. East Afr Med J. 2008 Oct;85(10):514-9
13. da Costa-Silva CM, Jeremias F, de Souza JF, Cordeiro Rde C, Santos-Pinto L, Zuanon AC. Molar incisor hypomineralization: prevalence, severity and clinical consequences in Brazilian children. Int J Paediatr Dent. 2010 Nov;20(6):426-34.
14. Zawaideh FI, Al-Jundi SH, Al-Jaljoli MH. Molar incisor hypomineralisation: prevalence in Jordanian children and clinical characteristics. Eur Arch Paediatr Dent. 2011 Feb;12(1):31-6.
15. Balmer R, Toumba J, Godson J, Duggal M. The prevalence of molar incisor hypomineralisation in Northern England and its relationship to socioeconomic status and water fluoridation. Int. J. Paediatr. Dent. 2012;22:250–257.
16. Negre-Barber A, Montiel-Company JM, Boronat-Catalá M, Catalá-Pizarro M, Almerich-Silla JM. Hypomineralized Second Primary Molars as Predictor of Molar Incisor Hypomineralization. Sci Rep. 2016 Aug 25;6:31929.
17. Tourino LF, Corrêa-Faria P, Ferreira RC, Bendo CB, Zarzar PM, Vale MP. Association between Molar Incisor Hypomineralization in Schoolchildren and Both Prenatal and Postnatal Factors: A Population-Based Study. PLoS One. 2016 Jun 9;11(6):e0156332.
18. Dantas-Neta NB, Moura LF, Cruz PF, Moura MS, Paiva SM, Martins CC, Lima MD. Impact of molar-incisor hypomineralization on oral health-related quality of life in schoolchildren. Braz Oral Res. 2016 Oct 24;30(1):e117.
19. Salem K, Aziz D, Asadi M. Prevalence and Predictors of Molar Incisor Hypomineralization (MIH) among Rural Children in Northern Iran. Iran J Public Health. 2016;45(11):1528-1530.
20. Al-Hammad, N. S., Al-Dhubaiban, M., Alhowaish, L., & Bello, L. L. (2018). Prevalence and Clinical Characteristics of Molar-Incisor-Hypomineralization in School Children in Riyadh, Saudi Arabia. International Journal of Medical Science and Clinical Invention, 5(3), 3570–3576..
21. Koruyucu M, Özel S, Tuna EB. Prevalence and etiology of molar-incisor hypomineralization (MIH) in the city of Istanbul. J Dent Sci. 2018;13(4):318-328.
22. Rizk H, Al-Mutairi MM, Habibullah MA. The prevalence of molar-incisor hypomineralization in primary schoolchildren aged 7–9 years in Qassim Region of Saudi Arabia. J Interdiscip Dentistry 2018;8:44-8.
23. Saitoh M, Nakamura Y, Hanasaki M, et al. Prevalence of molar incisor hypomineralization and regional differences throughout Japan. Environ Health Prev Med. 2018;23(1):55
24. Davenport M, Welles AD, Angelopoulou MV, et al. Prevalence of molar-incisor hypomineralization in Milwaukee, Wisconsin, USA: a pilot study. Clin Cosmet Investig Dent. 2019;11:109-117.
25. Amend, S. et al. Prevalence of molar-incisor-hypomineralisation (MIH) among 6–12-year-old children in Central Hesse (Germany). Clin. Oral Investig.25, 2093–2100 (2021).
26. Abdalla, H. E., Abuaffan, A. H. & Kemoli, A. M. Molar incisor hypomineralization, prevalence, pattern and distribution in Sudanese children. BMC Oral Health 21, 9 (2021).
27. Ilczuk-Rypuła, D.; Zalewska, M.; Pietraszewska, D.; Dybek, A.; Nitecka-Buchta, A.; Postek-Stefa ´ nska, L. Prevalence and Possible Etiological Factors of Molar-Incisor Hypomineralization (MIH) in Population of Silesian Children in Poland: A Pilot Retrospective Cohort Study. Int. J. Environ. Res. Public Health 2022, 19, 8697.
28. Damares Lago, Jéssica et al. "Molar-Incisor Hypomineralization: Prevalence Comparative Study in 6 Years of Interval." TheScientificWorldJournal vol. 2022 4743252. 9 Dec. 2022,
29. Parikh DR, Ganesh M, Bhaskar V. Prevalence and characteristics of Molar Incisor Hypomineralisation (MIH) in the child population residing in Gandhinagar, Gujarat, India. Eur Arch Paediatr Dent. 2012;13:21–6
30. ..H. T. Ajoy Rao, Dr. Sharan S Sargod; Prevalence and Sex Predilection of Molar Incisor Hypomineralization Among Children Aged 6–12 Years in Mangalore , Karnataka; Volume : IV, Issue : XI, November – 2014
31. Bhaskar SA, Hegde S; Molar-incisor hypomineralization: prevalence, severity and clinical characteristics in 8- to 13-year-old children of Udaipur, India. J Indian Soc Pedod Prev Dent. 2014 Oct-Dec;32(4):322-9.
32. Mittal R, Chandak S, Chandwani M, Singh P, Pimpale J. Assessment of association between molar incisor hypomineralization and hypomineralized second primary molar. J Int Soc Prev Community Dent. 2016 Jan-Feb;6(1):34-9.
33. Subramaniam P, Gupta T, Sharma A. Prevalence of molar incisor hypomineralization in 7–9-year-old children of Bengaluru City, India. Contemp Clin Dent 2016;7:11-5.
34. Yannam SD, Amarlal D, Rekha CV. Prevalence of molar incisor hypomineralization in school children aged 8-12 years in Chennai. J Indian Soc Pedod Prev Dent. 2016 Apr-Jun;34(2):134-8.
35. Mishra A, Pandey RK. Molar Incisor Hypomineralization: An Epidemiological Study with Prevalence and Etiological Factors in Indian Pediatric Population. Int J Clin Pediatr Dent. 2016;9(2):167-171.

36. Makne S.G.and Kakade A. Prevalence of molar incisor hypomineralisation in municipal school going children in Mumbai. Int J Res Health Sci 2017; 5(3): 13-17.
37. Rai A, Singh A, Menon I, Singh J, Rai V, Aswal GS. Molar Incisor Hypomineralization: Prevalence and Risk Factors Among 7-9 Years Old School Children in Muradnagar, Ghaziabad. Open Dent J. 2018;12:714-722.
38. Kirthiga M, Poornimal P, Praveen R, Gayathri P, Manju P, Priya M. Prevalence and severity of molar incisor hypomineralization in children aged 11-16 years of a city in Karnataka, Davangere. Journal of Indian Society of Pedodontics and Preventive Dentistry. 2015;33(3):213–217.
39. Devashish, Sanjeev Datana, SS Agarwal, SK Bhandari. Molar Incisor Hypomineralisation (MIH): Estimating prevalence and characteristics among children of Pune, Maharashtra. International Journal of Dental Research and Reviews, 2019, 2:11.
40. Padavala S, Sukumaran G. Molar incisor hypomineralization and its prevalence. Contemp Clin Dent 2018;9:S246-50.
41. Rai PM, Jain J, Raju AS, Nair RA, Shashidhar K, Dsouza S. Prevalence of Molar Incisor Hypomineralization among School Children Aged 9 to 12 Years in Virajpet, Karnataka, India. Open Access Maced J Med Sci. 2019;7(6):1042-1046.
42. Goswami M, Bhushan U, Pandiyan R, Sharma S. Molar Incisor Hypomineralization-An Emerging Burden: A Short Study on Prevalence and Clinical Characteristics in Central Delhi, India. Int J Clin Pediatr Dent. 2019;12(3):211-214. doi:10.5005/jp-journals-10005-1624
43. Peedikayil FC, Tomy NC, Chandru TP, Muthedath M, Jose J. Molar incisor hypomineralization in North Malabar: An epidemiological study. Dent Med Res 2019;7:40-4.
44. Emmatty TB, Eby A, Joseph MJ, Bijimole J, Kavita K, Asif I. The prevalence of molar incisor hypomineralization of schoolchildren in and around Muvattupuzha, Kerala. J Indian Soc Pedod Prev Dent 2020;38:14-9
45. Joby Peter, Vijai S., Krishna Kumar, Anaswara and Hannath Beevi (2020); prevalence of molar insisorhypomineralization of children in malappuram, kerala, india int. J. of adv. Res. 8 (dec). 922-928
46. Singh R, Srivastava B, Gupta N. Prevalence and pattern of molar incisor hypomineralization in Delhi region. J Anat Soc India 2020;69:150-4.
47. Khan A, Garg N, Mayall SS, et al. Prevalence, Pattern, and Severity of Molar Incisor Hypomineralization in 8–12-year-old Schoolchildren of Moradabad City. Int J Clin Pediatr Dent 2022;15(2):168–174.
48. Verma S, Dhinsa K, Tripathi AM, Saha S, Yadav G, Arora D. Molar Incisor Hypomineralization: Prevalence, Associated Risk Factors, Its Relation with Dental Caries and Various Enamel Surface Defects in 8-16-year-old Schoolchildren of Lucknow District. Int J Clin Pediatr Dent. 2022 Jan-Feb;15(1):1-8.
49. Prakrit Sharma, Sandeep Singh Siddhu, Tika Thapa. Assessing the Prevalence of Molar-Incisor Hypomineralisation among School Children in a North Indian District. IHRJ [Internet]. 2022Jul.31 [cited 2023Jan.6];6(4):OR1-OR3.
50. Alaluusua S. Aetiology of Molar-Incisor Hypomineralisation: A systematic review. Eur Arch Paediatr Dent. 2010;11(2):53-58. doi:10.1007/BF03262713
51. Alhowaish L, Baidas L, Aldhubaiban M, Bello LL, Al-Hammad N. Etiology of Molar-Incisor Hypomineralization (MIH): A Cross-Sectional Study of Saudi Children. Children (Basel). 2021 Jun 2;8(6):466.
52. DJ Reid, MC Dean. Journal of human evolution 50 (3), 329-346
53. William V, Messer LB, Burrow MF. Molar incisor hypomineralization: review and recommendations for clinical management. Pediatr Dent. 2006;28(3):224-32.
54. Kreshover SJ, Clough OW. Prenatal influences on tooth development. II. Artificially induced fever in rats. J Dent Res 1953;32(4):565-77.
55. Tsang RC, Donovan EF, Steichen JJ. Calcium physiology and pathology in the neonate. Pediatr Clin N Am 1976;23:611-26
56. Garg N, Jain AK, Saha S, Singh J. Essentiality of Early Diagnosis of Molar Incisor Hypomineralization in Children and Review of its Clinical Presentation, Etiology and Management. Int J Clin Pediatr Dent 2012;5(3):190-196.
57. Whatling R, Fearne JM. Molar incisor hypomineralisation: A study of aetiological factors in a group of UK children. Int J Paed Dent 2008;18:155-234
58. Muratbegovic A, Markovic N, Ganibegovic Selimovic M. Molar incisor hypomineralization in Bosnia and Herzegovina: aetiology and clinical consequences in medium caries activity population. Eur Arch Paediatr Dent. 2007;8(4):189-94
59. Jeremias F, Koruyucu M, Kuchler E C et al. Genes expressed in dental enamel development are associated with molar-incisor hypomineralization. Arch Oral Biol 2013; 58: 1434–1442.
60. Teixeira R J, Andrade N S, Queiroz L C et al. Exploring the association between genetic and environmental factors and molar incisor hypomineralization: evidence from a twin study. Int J Paediatr Dent 2018; 28: 198–206
61. Weerheijm KL, Duggal M, Mejàre I, Papagiannoulis L, Koch G, Martens LC, Hallonsten AL. Judgement criteria for molar incisor hypomineralisation (MIH) in epidemiologic studies: a summary of the European meeting on MIH held in Athens, 2003. Eur J Paediatr Dent. 2003 Sep;4(3):110-3.
62. Weerheijm KL. Molar-Incisor-Hypomineralisation (MIH). Eur J Paediatr Dent 2003;4:114-20.
63. Ghanim A, Elfrink M, Weerheijm K, Mariño R, Manton D. A practical method for use in epidemiological studies on enamel hypomineralisation. Eur Arch Paediatr Dent. 2015;16(3):235-246
64. Willmott N. Molar incisor hypomineralization. Dent Nursing. 2011;7(3):132-7.
65. William V, Messer LB, Burrow MF. Molar incisor hypomineralization: review and recommendations for clinical management. Pediatr Dent. 2006;28(3):224-32.
66. Lygidakis NA, Wong F, Jalevik B, Vierrou AM, Alaluusua S, Espelid I. Best Clinical Practice Guidance for clinicians dealing with children presenting with Molar-Incisor-Hypomineralization (MIH): An EAPD Policy Document. Eur Arch Paediatr Dent. 2010;11(2):75-81.
67. Willmott NS, Bryan RA, Duggal MS. Molar-incisor-hypomineralisation: a literature review. Eur Arch Paediatr Dent. 2008;9(4):172-179. doi:10.1007/BF03262633
68. William V, Messer LB, Burrow MF: Molar-incisor-hypomineralisation: Review and recommendations for clinical management. Paediatr Dent 2006;28(3):224-32
69. Fayle SA. Molar incisor hypomineralization: Restorative management. Eur J Paediatr Dent 2003;4:121-26.
70. Lygidakis NA, Chaliasou A, Siounas G. Evaluation of composite restoration in hypomineralised permanent molars: a four-year clinical study. Eur J Paediatr Dent 2003; 3: 143-148
71. Croll TP. Restorative options for malformed permanent molars in children. Compend Contin Educ Dent. 2000;21:676-682
72. Mathu-Muju K, Wright JT. Diagnosis and treatment of molar incisor hypomineralisation. Compend Contin Educ Dent 2006;27(11):604-10
73. Zagdwon AM, Fayle SA, Pollard MA. A prospective clinical trial comparing preformed metal crowns and cast restorations for defective first permanent molars. Eur J Paediatr Dent. 2003;4(3):138-142.
74. Alfarraj JH, Alsaeed AA. Clinical Management of Molar Incisor Hypomineralization Affected Molars in a Pediatric Patient Including Endodontic Treatment, Case Report and Review of the Literature. Clin Cosmet Investig Dent. 2022 Jun 28;14:183-189.
75. Williams JK, Gowans AJ. Hypomineralised first permanent molars and the orthodontist. Eur J Paediatr Dent 2003;4:129-132.

Summary of epidemiological studies of MIH **(Table 1)**

Year	Country	Author	Study Group	Sample size	MIH prevalence
2001	Finland	Leppaniemi et al[9]	7-13 yrs	488	19.3%
2001	Netherland	Weerheijm et al[1]	11 yrs	497	9.7%
2002	UK	Zagdwon et al[10]	7 yrs	307	14.6%
2007	Germany	Preusser et al[11]	6-12 yrs	1022	5.9%
2008	Kenya	Kemoli et al[12]	6-8 yrs	3591	13.77%
2010	Brazil	de.Costo.Silva et al[13]	6-12 yrs	918	19.8%
2011	Jordon	Zawaideh et al[14]	7-9 yrs	3666	17.6%
2011	Northern England	Balmer et al[15]	12 yrs	3233	15.9%
2016	Spain	Negre-Barber et al[16]	8-9yrs	414	24.15%
2016	Brazil	Tourino et al[17]	8-9yrs	1181	20.41%
2016	Brazil	Dantas-Neta et al.[18]	9-11yrs	594	18.35%
2016	Iran	Katayoun Salem et al[19]	6-13 yra	553	18.4%
2018	Saudi Arabia	Nou S. Al- Hammad et al[20]	8-10 yrs	924	64.6%
2018	Turkey	Mine Koruyucu et al[21]	8- yrs	1511	14.2%
2018	Saudi Arabia	Hazim Rizk et al[22]	7-9 yrs	411	25.1%
2018	Japan	Masato Satioh et al[23]	7-9 yrs	4496	19.8%
2019	USA	Davenport et al[24]	7-12 yrs	375	9.6%
2020	Germany	Amend et al[25]	8-10yrs	1820	65.2%
2021	Sudan	Abdalla et al[26]	8-11 yrs	470	12.5%
2022	Silesia	Rypula et al[27]	7.1-10.9 yrs	613	6.2%
2022	Brazil	Jessica Damares Lago et al[28]	6-12 yrs	545	14.3%

Indian survey **(Table 2)**

2012	India	Parikh De et al[29]	8-12 yrs	1366	9.2%
2014	India	HT Ajay Rao et al[30]	6-12 yrs	250	17.2%
2014	India	Bhaskar et al[31]	8-13 yrs	1173	9.2%
2016	India	Mittal[32]	12-16 yrs	1726	13.21%
2016	India	Subhramanium et al[33]	7-9yrs	2500	0.48%
2016	India	Yannam et al[34]	8-12 yrs	2864	9.67%
2016	India	Mishra et al[35]	8-12 yrs	1369	13.9%
2017	India	Makne et al[36]	7-0 yrs	544	7.90%
2018	India	Rai et al[37]	7-9 yrs	992	21.4%
2019	India	Kirthiga et al[38]	11-16 yrs	2000	8.9%
2019	India	Devashish D et al[39]	8-12 yrs	1080	8.3%
2019	India	Padvala et al[40]	7-12 yrs	170	12.9%
2019	India	Rai et al[41]	9-2 yrs	1600	13.12%
2019	India	Goswami et al[42]	6-12 yrs	1026	1.17%
2019	India	Peedikayil et al[43]	6-10 yrs	2000	19.8%
2019	India	Emmaty et al[44]	8-15 yrs	5318	4.1%
2020	India	Peter et al[45]	6-12 yrs	2000	6.75%
2020	India	Singh et al[46]	7-10 yrs	649	15%
2022	India	Khan et al[47]	8-12 yrs	2300	3.96%
2022	India	Verma et al[48]	8-16 yrs	5585	7.6%
2022	India	Sharma et al[49]	8-12 yrs	793	23.5%

Table 3: Modified DDE index (FDI 1992[8])

Mild	<30% of the tooth's enamel surface area visibly disrupted(this encompasses the entire range reported in most other studies)
Moderate	31-49% of the tooth's enamel surface area visibly disrupted
Severe	>50% of the tooth's enamel surface area visibly disrupted

Table 4: Definitions of criteria used for diagnosing MIH (Weerheijm et al 2001[62])

Criteria	Definitions
Opacity	A defect involving an alteration in the translucency of the enamel, variable in degree. The defective enamel is of normal thickness with a smooth surface and can be white, yellow or brown in color. The border of the lesions is demarcated.
PEB	A defect that indicated deficiency of the surface after eruption of tooth. This may be caused by such factors as trauma and attrition.
Atypical restoration	Size and shape of restoration do not conform to typical restorative characteristics. In most cases restorations will be extended to the buccal or the palatinal smooth surface. At the border of the restoration opacity may be noticed.
Extraction due to MIH	Absence of a molar should be related to the other tooth of the dentition. Absence of a first permanent molar in a sound dentition is suspected to have been an MIH.

Table 5: EAPD scoring criteria for MIH (Ghanim et al.2011[63])

Code	Criteria
0	Enamel defects free
1	White / creamy demarcated opacities, no PEB
1a	White / creamy demarcated opacities, with PBE
2	Yellow / brown demarcated opacities, no PEB
2a	Yellow / brown demarcated opacities, with PEB
3	Atypical restoration
4	Missing because of MIH
5	Partially erupted (i.e .,less than one third of crown high) with evidence of MIH
6	Unerupted / partially erupted with no evidence of MIH
7	Diffuse opacities (not MIH)
8	Hypoplasia (not MIH)
9	Combined lesion (diffuse opacities/ hypoplasia with MIH)
10	Demarcated opacities incisors only