ACTA SCIENTIFIC DENTAL SCIENCES (ISSN: 2581-4893)

Volume 3 Issue 6 June 2019

Research Article

Molar Incisor Hypomineralisation – An Overview

Narmatha M[1]*, Prathima GS[2] and Selvabalaji A[3]

[1]*Department of Paedodontics and Preventive Dentistry, Indira Gandhi Institute of Dental Sciences, Sri Balaji Vidyapeeth (Deemed to be University), Puducherry, India*

[2]*Professor and Head , Department of Paedodontics and Preventive Dentistry, Indira Gandhi Institute of Dental Sciences, Sri Balaji Vidyapeeth (Deemed to be University), Puducherry, India*

[3]*Reader, Department of Paedodontics and Preventive Dentistry, Indira Gandhi Institute of Dental Sciences, Sri Balaji Vidyapeeth, Deemed to be University, Puducherry, India*

***Corresponding Author:** Narmatha M, Post Graduate Scholar, Department of Paedodontics and Preventive Dentistry, Indira Gandhi Institute of Dental Sciences, Sri Balaji Vidyapeeth (Deemed to be University), Puducherry, India.

Received: April 03, 2019; **Published:** May 17, 2019

DOI: 10.31080/ASDS.2019.03.0544

Abstract

Molar Incisor Hypomineralisation is one of the common developmental defects of enamel in children. The aetiology of this defect is unknown, but some possible risk factors have been associated. The clinical features range from white/yellow/brown demarcated opacities to enamel disintegration. Due to its characteristic features, its clinical management is challenging to the Dentists. The treatment modalities for Molar Incisor Hypomineralisation can either be preventive/restorative/ surgical, based on the severity of the defect. The aim of this review article is to discuss about the prevalence, aetiology, clinical features, diagnosis and management of Molar Incisor Hypomineralisation condition.

Keywords: Aetiology; Enamel Defect; Prevalence; Management; Risk Factors

Abbreviations

MIH: Molar Incisor Hypomineralisation; HSPM: Hypomineralised Second Primary Molar.

Introduction

In human body, Dental enamel is found to be the hardest tissue comprising of 98% mineral and less than 2% organic matrix and water [1]. Dental enamel is sensitive to environmental disturbances during development which results in permanent variations of tooth enamel, because of its non-remodeling nature [2]. In both the primary and permanent dentition, enamel defects of developmental origin are more common. The defects can be divided into enamel hypoplasia and enamel hypomineralisation. Enamel hypoplasia is a quantitative defect whereas enamel hypomineralisation is a qualitative defect characterized by abnormal translucency of enamel with a well-defined border, variable in degree and is white, yellow or brown in colour with demarcated opacity [3] . These defects were unique as it affects only First Permanent Molars and Permanent Incisors but sometimes it is also seen in other teeth such as Permanent canines and Second deciduous molars. It was later termed as "Molar Incisor Hypomineralisation" by Weerheijm in 2001 [4].

In MIH teeth, the enamel will be porous which makes it hypersensitive and prone to dental caries [5]. It is not only annoying to children but also to the Dentists as the clinical management will be difficult due to sensitivity and failure of achieving analgesia [6]. This problem can be overcome by treating the lesions earlier. A thorough knowledge regarding MIH is necessary for the clinician for the early management of such defects [7]. Hence in this review article, the prevalence, aetiology, clinical features, diagnosis and management of Molar Incisor Hypomineralisation will be discussed.

Materials and Methods

An electronic database search was done using PUBMED and Google scholar search engines to select the articles for this review. The keywords used were Aetiology, Molar incisor hypomineralisation, Prevalence, Management, Risk factors.

Results and Discussion

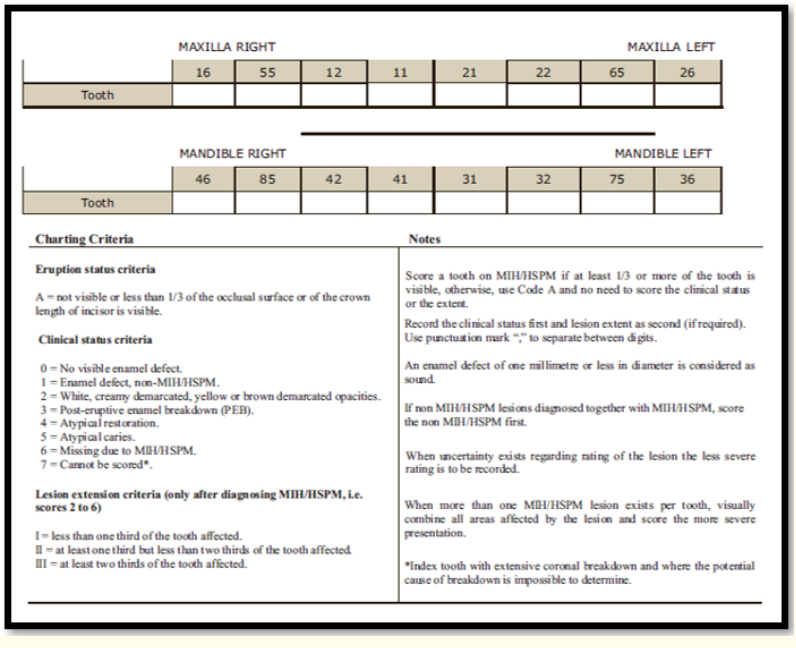
Background of MIH:

Molar Incisor Hypomineralisation (MIH) was noticed as early as in the 17th -18th century following an examination of archaeological sub-adult samples of London cemetery [8]. The first epidemiologi-

cal studies evaluating Molar Incisor Hypomineralisation was by Koch in Sweden in the 1970's. He termed it as "idiopathic enamel hypomineralisation" [9].

Subsequently, different terms were used for the same condition such as "hypomineralised permanent first molars", "non-fluoride hypomineralisation in permanent first molars", "cheese molars", "Internal enamel hypoplasia", "non-endemic mottling of enamel", "opaque spots", "idiopathic enamel opacities" and "enamel opacities". Finally, "Molar Incisor Hypomineralisation" term was coined in 2001 by European Academy of Paediatric Dentistry at the 5th congress meeting by Weerheijm., *et al*. MIH is defined as "a hypomineralisation of systemic origin of one to four permanent first molars frequently associated with affected incisors" [4]. The judgemental criteria for diagnosing Molar Incisor Hypomineralisation teeth was given by Weerheijm., *et al*. in 6th EAPD congress meeting at Athens in 2003. The judgemental criteria includes Demarcated opacities (white/yellow/brown in colour), Post-eruptive breakdown of enamel (loss of tooth structure after eruption and is commonly present with the pre-existing demarcated opacities), Atypical restorations (restorations are extended buccally or palatally due to gross breakdown of enamel and in restored teeth, opacities are present at the border. In incisors the restoration is seen extending labially with no relevant history of trauma), Extracted MIH affected molars (it is judged by the presence of any white/yellow/brown demarcated opacities or atypical restorations seen in the other First Permanent Molars and also demarcated opacities on the incisors which is suspected for MIH) and the Failure of molars or incisors eruption due to MIH [5]. Lygidakis N., *et al*. (2010) published a document regarding the diagnosis, treatment planning of MIH. He recommended that demarcated opacities less than 1mm should not be considered as MIH and classified the type of severity as mild/moderate and severe. There is no accurate treatment for MIH affected teeth but preventive or restorative treatment could be the best possible option [7].

Elfrink M.E.C., *et al*. (2015) proposed a Standardisation protocol for upcoming MIH and HSPM prevalence and aetiological studies and he recommended that minimum of 300 sample subjects are required for prevalence studies and minimum of 1000 sample subjects are needed for aetiology studies. 8 years of age is ideal for the clinical examination for MIH and 5 years of age for HSPM [10]. Ghanim A., *et al*. (2015) published a document in EAPD explaining grading method for hypomineralisation and other enamel defects similar to MIH. He proposed a short charting form for simple screening surveys and a long charting form for prospective, longitudinal observational studies (Figure 1, 2) [11].



	MAXILLA RIGHT						MAXILLA LEFT	
Tooth	16	55	12	11	21	22	65	26

	MANDIBLE RIGHT						MANDIBLE LEFT	
Tooth	46	85	42	41	31	32	75	36

Charting Criteria

Eruption status criteria

A = not visible or less than 1/3 of the occlusal surface or of the crown length of incisor is visible.

Clinical status criteria

0 = No visible enamel defect.
1 = Enamel defect, non-MIH/HSPM.
2 = White, creamy demarcated, yellow or brown demarcated opacities.
3 = Post-eruptive enamel breakdown (PEB).
4 = Atypical restoration.
5 = Atypical caries.
6 = Missing due to MIH/HSPM.
7 = Cannot be scored*.

Lesion extension criteria (only after diagnosing MIH/HSPM, i.e. scores 2 to 6)

I = less than one third of the tooth affected.
II = at least one third but less than two thirds of the tooth affected.
III = at least two thirds of the tooth affected.

Notes

Score a tooth on MIH/HSPM if at least 1/3 or more of the tooth is visible, otherwise, use Code A and no need to score the clinical status or the extent.

Record the clinical status first and lesion extent as second (if required). Use punctuation mark "," to separate between digits.

An enamel defect of one millimetre or less in diameter is considered as sound.

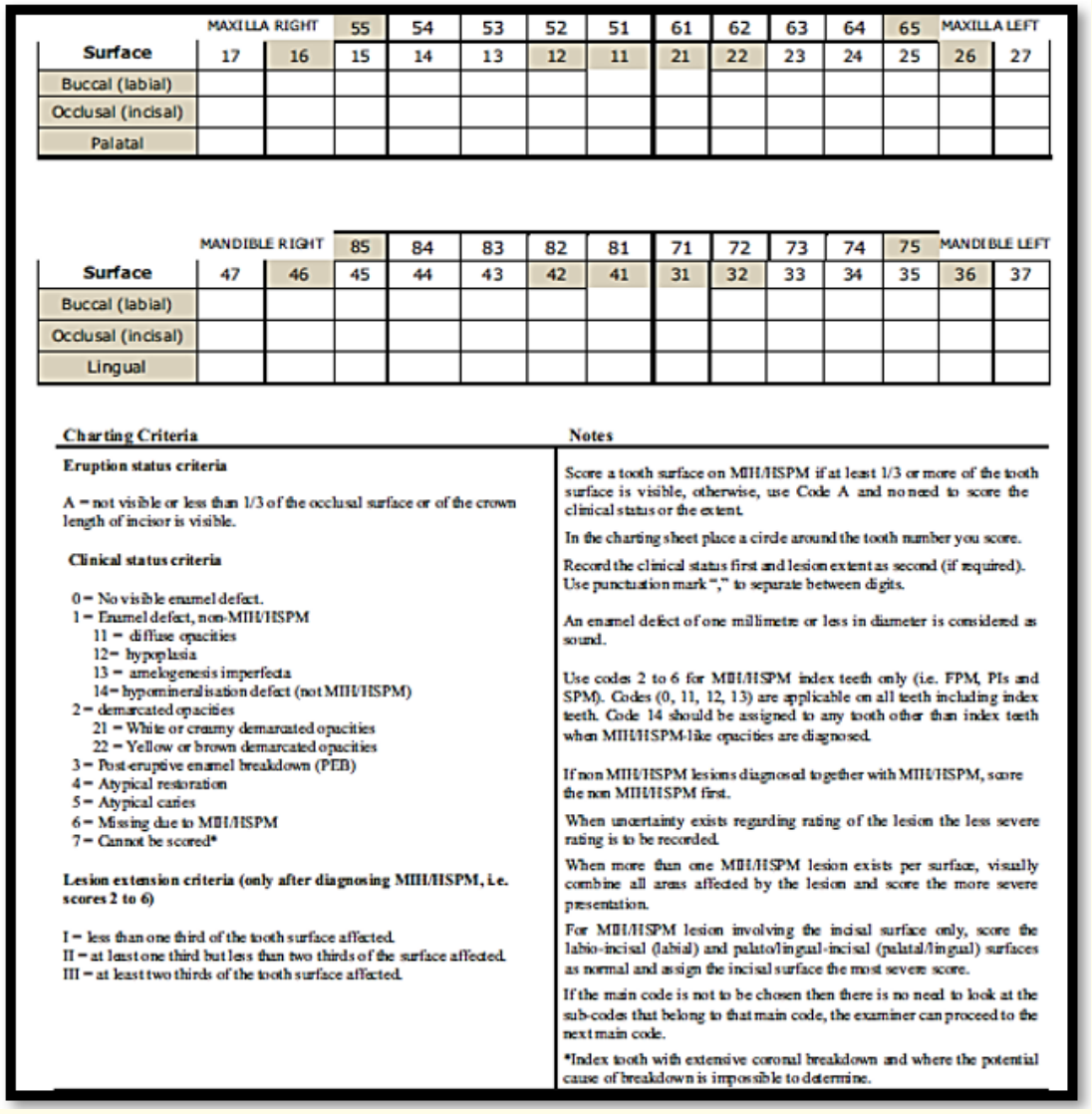
If non MIH/HSPM lesions diagnosed together with MIH/HSPM, score the non MIH/HSPM first.

When uncertainty exists regarding rating of the lesion the less severe rating is to be recorded.

When more than one MIH/HSPM lesion exists per tooth, visually combine all areas affected by the lesion and score the more severe presentation.

*Index tooth with extensive coronal breakdown and where the potential cause of breakdown is impossible to determine.

Figure 1: MIH/HSPM clinical data recording sheet- First Permanent Molars, Permanent Incisors and Second Primary Molars (short form).
Source: Ghanim A, Elfrink M, Weerheijm K, Marino R, Manton D. A practical method for use in epidemiological studies on enamel hypomineralisation. Eur Arch Paediatr Dent. 2015; 16:235–46.



	MAXILLA RIGHT		55	54	53	52	51	61	62	63	64	65	MAXILLA LEFT	
Surface	17	16	15	14	13	12	11	21	22	23	24	25	26	27
Buccal (labial)														
Occlusal (incisal)														
Palatal														

	MANDIBLE RIGHT		85	84	83	82	81	71	72	73	74	75	MANDIBLE LEFT	
Surface	47	46	45	44	43	42	41	31	32	33	34	35	36	37
Buccal (labial)														
Occlusal (incisal)														
Lingual														

Charting Criteria

Eruption status criteria

A = not visible or less than 1/3 of the occlusal surface or of the crown length of incisor is visible.

Clinical status criteria

0 = No visible enamel defect.
1 = Enamel defect, non-MIH/HSPM
  11 = diffuse opacities
  12= hypoplasia
  13 = amelogenesis imperfecta
  14= hypomineralisation defect (not MIH/HSPM)
2 = demarcated opacities
  21 = White or creamy demarcated opacities
  22 = Yellow or brown demarcated opacities
3 = Post-eruptive enamel breakdown (PEB)
4 = Atypical restoration
5 = Atypical caries
6 = Missing due to MIH/HSPM
7 = Cannot be scored*

Lesion extension criteria (only after diagnosing MIH/HSPM, i.e. scores 2 to 6)

I = less than one third of the tooth surface affected.
II = at least one third but less than two thirds of the surface affected.
III = at least two thirds of the tooth surface affected.

Notes

Score a tooth surface on MIH/HSPM if at least 1/3 or more of the tooth surface is visible, otherwise, use Code A and no need to score the clinical status or the extent.

In the charting sheet place a circle around the tooth number you score.

Record the clinical status first and lesion extent as second (if required). Use punctuation mark "," to separate between digits.

An enamel defect of one millimetre or less in diameter is considered as sound.

Use codes 2 to 6 for MIH/HSPM index teeth only (i.e. FPM, PIs and SPM). Codes (0, 11, 12, 13) are applicable on all teeth including index teeth. Code 14 should be assigned to any tooth other than index teeth when MIH/HSPM-like opacities are diagnosed.

If non MIH/HSPM lesions diagnosed together with MIH/HSPM, score the non MIH/HSPM first.

When uncertainty exists regarding rating of the lesion the less severe rating is to be recorded.

When more than one MIH/HSPM lesion exists per surface, visually combine all areas affected by the lesion and score the more severe presentation.

For MIH/HSPM lesion involving the incisal surface only, score the labio-incisal (labial) and palato/lingual-incisal (palatal/lingual) surfaces as normal and assign the incisal surface the most severe score.

If the main code is not to be chosen then there is no need to look at the sub-codes that belong to that main code, the examiner can proceed to the next main code.

*Index tooth with extensive coronal breakdown and where the potential cause of breakdown is impossible to determine.

Figure 2: MIH/HSPM clinical data recording sheet- First Permanent Molars, Permanent Incisors and Second Primary Molars (long form).
Source: Ghanim A, Elfrink M, Weerheijm K, Marino R, Manton D. A practical method for use in epidemiological studies on enamel hypomineralisation. Eur Arch Paediatr Dent. 2015; 16:235–46.

Prevalence

Molar Incisor hypomineralisation prevalence has been reported with a wide variation of about 2.4 to 40.2% around the world. The lowest prevalence (2.5%) of MIH was observed among Chinese children and the highest (40.2%) prevalence was reported in Brazil (Table 1) [10,12-17]. In India, the first study was conducted in Gandhinagar where the prevalence was 9.8%.The prevalence in South India cities such as Chennai, Salem, Bangalore, Tiruchengode, it was reported to be 9.8% , 7.2% , 0.48% , 5.25% respectively (Table 2) [10,18,19]. This wide variation is because of alteration in population, genetics, environment and socio-economic status of the population [20].

Study	Country	Sample size	Sample population age	Criteria followed	Prevalence (%)
Koch., *et al*. 1987	Sweden	2226	9-13 years old	Koch., *et al*. 1987	3.6- 15.4
Alaluusua., *et al*. 1996	Finland	97	12 years	Alaluusua 1996	25
Alaluusua., *et al*. 1996	Finland	102	6-7 years	Alaluusua 1996	17
Jalevik., *et al*. 2001	Sweeden	516	7-8 years	mDDE	18.4
Leppaniemi., *et al*. 2001	Finland	488	7-13 years	Alaluusua 1996	19.3
Weerheijm., *et al*. 2001	The Netherlands	497	11 years	Weerheijm 2001	9.7
Dietrich., *et al*. 2003	Germany	2408	10-17 years	Jalevik 2001	5.6
Calderara., *et al*. 2005	Italy	227	7-8 years	Calderara., *et al*. 2005	13.7
Fteita., *et al*. 2006	Libya	378	7-9 years	Ns	2.9
Preusser., *et al*. 2007	Germany	1002	6-12 years	Koch., *et al*. 1987	5.9
Jasulaityte., *et al*. 2007	Lithuania	442	9 years	EAPD	9.7
Muratbegovic., *et al*. 2007	Bosnia- Herzegovina	560	12 years	EAPD	12.3
Jasulaityte., *et al*. 2008	The Netherlands	1157	6-12 years	Weerheijm 2001	14.3
Kemloli 2008	Kenya	3591	6-8 years	Kemoli 2008	13.7
Kukleva., *et al*. 2008	Bulgaria	2960	7-14 years	EAPD	3.6 (2.4-7.8)
Kuscu., *et al*. 2008	Turkey	147	7-9 years	EAPD	14.9
Lygidakis., *et al*. 2008	Greece	3518	5-12	EAPD	10.2
Wogelius., *et al*. 2008	Denmark	745	6-8 years	EAPD	37.3
Cho., *et al*. 2008	China	2635	11-14 years	EAPD	2.8
Kuscu., *et al*. 2009	Turkey	197	7-10 years	EAPD	9.1/9.2
Mahoney and Morrison., *et al*. 2009	New Zealand	522	7-10 years	mDDE	14.9
Soviero., *et al*. 2009	Brazil	249	7-13 years	EAPD	40.2
Costa-Silva., *et al*. 2010	Brazil	918	6-12 years	EAPD	19.8
Mahoney and Morrison., *et al*. 2011	New Zealand	234	7-10 years	mDDE& EAPD	18.8/15.7
Zawaideh., *et al*. 2011	Jordan	3241	8.4 years	EAPD	17.6
Biondi., *et al*. 2011	Argentina	1098	11.3 years	DDE	15.9
Ghanim., *et al*. 2011	Iraq	823	7-9 years	EAPD	18.6
Ahmadi., *et al*. 2012	Iran	433	7-9 years	DDE & EAPD	12.7
Balmer., *et al*. 2012	Great Britain	3233	12 years	mDDE	15.9
Condo., *et al*. 2012	Italy	227	7-8 years	EAPD	7.3
Elfrink., *et al*. 2012	The Netherlands	6161	6 years	EAPD	8.7

Biondi., *et al*. 2012	Argentina	512	11.6 years	Mathu-Muju and Wright 2006	6.4
Martinez Gomez., *et al*. 2012	Spain	505	6-14 years	EAPD	17.8
Souza., *et al*. 2012	Brazil	903	6-12 years	EAPD	19.8
Souza., *et al*. 2013	Brazil	1151	7-12 years	EAPD	12.3
Ghanim., *et al*. 2013a	Iran	810	9-11 years	EAPD	20.2
Groselj and Jan 2013	Slovenia	478	6-11.5 years	mDDE/FDI & EAPD	21.4
Heitmuller., *et al*. 2013	Germany	1277	7-9 years	EAPD	9.7
Jeremias., *et al*. 2013	Brazil	1157	6-12 years	EAPD	12.3
Kohlboeck., *et al*. 2013	Germany	1126	10 years	EAPD	13.7
Sonmez., *et al*. 2013	Turkey	4049	7-12 years	EAPD	7.7
Allazzam., *et al*. 2014	Saudi Arabia	267	8-12 years	EAPD	8.6
Jankovic., *et al*. 2014	Bosnia- Herzegovina	141	8 years	EAPD	12.8
Kuhnisch., *et al*. 2014	Germany	693	10 years	EAPD	14.7
Petrou., *et al*. 2014	Germany	2395	8.1 years	EAPD	10.1
Pitphat., *et al*. 2014	Thailand	282	7-8 years	EAPD	27.7
Shrestha., *et al*. 2014	Nepal	749	7-12 years	EAPD	13.7
Wuollet., *et al*. 2014	Finland	818	7-13 years	EAPD	17.1
Nj., *et al*. 2014	Singapore	1083	7.7 years	EAPD	12.5
Garcia – Margarait., *et al*. 2014	Spain	840	8 years	EAPD	21.8
Kevrekidou., *et al*. 2015	Greece	1179&1156	8 years & 14 years	EAPD	21
Tourino., *et al*. 2016	Brazil	1181	8-9 years	EAPD	20.4
Negre-barber., *et al*. 2016	Spain	414	8-9 years	EAPD	24.2
Americano., *et al*. 2016	Brazil	469	7-11 years	EAPD	5.89
Gurrusquieta., *et al*. 2017	Mexico	582	6-12 years	EAPD	15.8
Saber., *et al*. 2018	Egypt	1001	8-12 years	Ghanim short charting criteria	2.8
Dantas-Neta., *et al*. 2018	Piaui, Brazil	558	8-10 years	EAPD	19.5
Koruyucu., *et al*. 2018	Istanbul	1511	8-11 years	EAPD	14.2
Saitoh M., *et al*. 2018	Japan	4496	7-9 years	EAPD	19.8

Table 1: Global Prevalence of MIH.

Study	Localities	Sample size	Sample population age	Criteria followed	Prevalence (%)
Parikh., *et al*. 2012	Gandhinagar, Gujarat	1366	8-12 years	EAPD	9.2
Mittal., *et al*. 2014	Chandigarh	1792	6-9 years	EAPD	6.3
Bhaskar and Hegde 2014	Udaipur, Rajasthan	1173	8-13 years	EAPD	9.5
Krishnan., *et al*. 2015	Salem, Tamilnadu	5000	9-14 years	EAPD	7.3
Kirthiga., *et al*. 2015	Davengere, Karnataka	2000	11-16 years	Cho., *et al*. criteria 2008	8.9
Tadikonda., *et al*. 2015	Udupi, Karnataka	352	11-15 years	EAPD	27
Siddaiah., *et al*. 2016	South Bangalore, Karnataka	1004	8-9 years	EAPD	11.5
Subramaniam., *et al*. 2016	Bangalore, Karnataka	2500	7-9 years	EAPD	0.48
Yannam SD., *et al*. 2016	Chennai, Tamilnadu	2864	8-12 years	EAPD	9.7
Mishra A., *et al*. 2016	Lucknow, Uttar Pradesh	1369	8-12 years	EAPD	13.9
Mittal., *et al*. 2016	Nagpur, Maharastra	1109	6-12 years	EAPD	7.11
Samuel., *et al*. 2017	Tiruchengode, Tamilnadu	4495	8-12 years	EAPD	5.25
Rai., *et al*. 2018	Ghaziabad	4020	7-9 years	mDDE	21.4

Table 2: Prevalence of MIH in India

Aetiology

The exact aetiology of MIH is unknown. To understand the possible aetiological factors of MIH it is important to remember the critical period i.e between 28th week of in utero life to the first 10 days of life after birth because, amelogenesis of the first permanent molars, permanent incisors and second primary molars begin at that time [3]. When any risk factor ensues during this intersecting period, hypomineralisation will occur in both the deciduous and permanent dentition. Second Deciduous Molars is termed as "Hypomineralised Second Primary Molar" (HSPM) by Elfrink., *et al*. in 2008 [21] and HSPM might be used as a predictor for "Molar Incisor Hypomineralisation" [22,23]. The prevalence of HSPM is reported to be 2.7-21.8% globally (Table 3) [10,24-26].

Study	Country	Sample size	Sample population age	Criteria followed	Prevalence (%)
Elfrink., *et al*. 2008	The Netherlands	386	5	EAPD	4.9
Elfrink., *et al*. 2009	The Netherlands	62	5	EAPD	21.8
Elfrink., *et al*. 2012	The Netherlands	6161	6	EAPD	9
Elfrink., *et al*. 2013	The Netherlands	6690	6	EAPD	9
Ghanim., *et al*. 2013	Iraq	809	7-9	EAPD	6.6
Costa Silva., *et al*. 2014	Brazil	134	4-6	EAPD	20.14
Kar., *et al*. 2014	India	308	3-5	mDDE	0
Kuhnisch., *et al*. 2014	Germany	693	10	EAPD	4
Ng., *et al*. 2014	Singapore	1083	7.7	EAPD	2.9
Mittal., *et al*. 2015	Uttar Pradesh	978	6-8	EAPD	5.6
Negre- Barber., *et al*. 2016	Spain	414	8-9	EAPD	14.5
Owen., *et al*. 2018	Australia	623	5	EAPD	14.1

Table 3: Global Prevalence of HSPM.

Ameloblasts are very sensitive, if the functioning of ameloblasts are disturbed either temporarily or permanently, it results in enamel hypoplasia or hypomineralisation which is mainly dependent on the time of insult [3]. Abnormal oxygen levels and respiratory acidosis which occur as a result of any respiratory or breathing diseases will alter the enamel pH level and so the growth of hydroxyapatite crystals will be retarded because of the inhibition of proteolytic enzyme activity. Ameloblastic function itself might be additionally affected by low oxygen levels found during the birth of preterm children [27,28]. Hypomineralisation of enamel might occur due to low level of calcium and phosphorous ions which leads to reduced deposition of calcium in the enamel [3]. Many retrospective clinical studies are conducted to evaluate the possible medical conditions associated with MIH. Retrospective studies have the source of information, mainly as the answer from parents regarding medical information of the child which include prenatal/ perinatal and postnatal medical history of the child [3,29]. Taking into consideration, all these factors into account, it is believed that prospective studies need to be done to arrive at accurate information about the possible aetiological factors of MIH. Several retrospective, cohort, case-control studies were performed and reported on the possible aetiological factors of "Molar Incisor Hypomineralisation" [29]. They are grouped into three factors as prenatal, perinatal and postnatal factors. The most common factors include maternal systemic illness, infections during pregnancy, Premature birth, caesarean type of delivery, hypoxia, hypocalcaemia and early childhood illness which occurs before three years of age [3,29].

Clinical features

- The hypomineralized enamel of affected molars and incisors will be soft, porous and have a discoloured chalky appearance
- Demarcated white/yellow/brown opacities usually limited to incisal or cuspal one third, rarely involving cervical one third
- Post eruptive enamel breakdown is common in molars due to occlusal loading and is not common in incisors
- Rapid caries progression due to bacterial penetration through porous enamel into subjacent dentin [6].

Signs

- Hypersensitivity during brushing is the most common sign and is due to porous enamel which leads to subclinical pulpal inflammation [6].

Differential diagnosis

It includes, Amelogenesis Imperfecta, Hypoplasia, Dental fluorosis, White spot lesions, Tetracycline staining, Erosion, White cuspal and marginal ridges and Turner's tooth [5].

Microscopic features

Microscopic analysis shows that MIH teeth have less prism structure with loosely packed apatite crystals and wider sheath regions [30].

Treatment approaches

A six step approach for clinical management of Molar Incisor Hypomineralisation was proposed by William., et al. in 2006.

It includes

1. Risk identification
2. Early diagnosis
3. Remineralization and desensitization
4. Prevention of caries and posteruption breakdown
5. Restorations and extractions
6. Maintenance [31].

The treatment decision for MIH is dependent on a number of factors. They are

- Patient's dental age
- Severity of the affected tooth
- Socio-economic status of the child [7].

Prevention

Prevention is the important treatment option during the early age because the affected tooth is more prone to dental caries and post eruptive breakdown. The different preventive measures include

- Dietary modification
- Using Desensitizing toothpaste
- Topical application of CPP-ACP with a cotton bud daily [31].
- Topical fluoride application
- Use of fluoridated tooth paste containing 1000 ppm fluoride [32].
- Use of CPP-ACP containing chewing gums [33].
- Glass ionomer cement (GIC) sealants which provide caries protection and reduce surface permeability
- Composite resin sealants [7] .

Interim treatment

- GIC cement [31].

Aesthetic treatment

- Resin infiltration [34].
- Microabrasion, bleach and sealant for anterior teeth: It can be treated by "Micro abrasion" with 18% hydrochloric acid or 37.5% phosphoric acid and abrasive paste. "Yellow or brownish-yellow opacities" are more porous and should be treated by bleaching with carbamide peroxide [35].

Restorative treatment

Adhesive materials are the most commonly preferred material as it adheres chemically with the tooth surface. It is not recommended in occlusal stress bearing areas and can be given only as intermittent restoration if required (poor moisture control and lack of cooperation of the child) and later should be replaced by definitive restoration with composite [7].

Resin composites are aesthetic materials having high wear resistance and adhesion properties. So they can be used either alone or in combination with GIC using a sandwich technique. Self-etching adhesive (SEA) was found to have better bond strength to MIH affected enamel than all-etch single-bottle adhesive (SBA) [31].

Preformed Stainless Steel Crowns are the treatment of choice in case of post eruptive breakdown in MIH teeth to provide full coverage to defective molars [31].

Extraction and orthodontic management:

Extraction should be carried out at an age of 8.5-9 years. At this age the second molars will be allowed to erupt in the First Permanent Molar region and thus achieve acceptable molar occlusion [36].

Conclusion

The prevalence of "Molar Incisor Hypomineralisation" seems to be increasing in children all around the world. There are high chances of misdiagnosing MIH affected teeth in adults as it gets masked by caries. Therefore early diagnosis of "Molar Incisor Hypomineralisation" is necessary, so that preventive measures can be

taken to prevent further complications. Parents and health professionals have to be educated on MIH, making them aware of the rising problem so that preventive measures can be taken as early as possible.

Acknowledgements

Nil

Conflict of interest

None

Bibliography

1. Seow WK. "Developmental defects of enamel and dentine: challenges for basic science research and clinical management". *Australian Dental Journal* 59 (2014): 143-154.

2. Sadashivamurthy Prashanth and Seema Deshmukh. "Missing links of molar incisor hypomineralization: a review". *Journal of International Oral Health* 4.1 (2012): 1-10.

3. Alaluusua S. "Aetiology of molar-incisor hypomineralisation: a systematic review". *European Archives of Paediatric Dentistry* 11.2 (2010): 53-58.

4. Weerheijm KL., *et al*. "Molar-incisor hypomineralisation". *Caries Research* 35.5 (2001): 390.

5. Weerheijm KL. "Molar incisor hypomineralisation (MIH)". *European Journal of Paediatric Dentistry* 4 (2003): 115-120.

6. Weerheijm Karin L. "Molar incisor hypomineralization (MIH): clinical presentation, aetiology and management". *Dental update* 31.1 (2004): 9-12.

7. Lygidakis NA., *et al*. "Best Clinical Practice Guidance for clinicians dealing with children presenting with Molar-Incisor-Hypomineralisation (MIH)". *European Archives of Paediatric Dentistry* 11.2 (2010): 75-81.

8. Ogden Alan R., *et al*. "Gross enamel hypoplasia in molars from subadults in a 16th–18th century London graveyard". *American Journal of Physical Anthropology* 133.3 (2007): 957-966.

9. Koch Göran., *et al*. "Epidemiologic study of idiopathic enamel hypomineralization in permanent teeth of Swedish children". *Community Dentistry and Oral Epidemiology* 15.5 (1987): 279-285.

10. Elfrink ME. C., *et al*. "Standardised studies on molar incisor hypomineralisation (MIH) and hypomineralised second primary molars (HSPM): a need". *European Archives of Paediatric Dentistry* 16.3 (2015): 247-255.

11. Ghanim A., *et al*. "A practical method for use in epidemiological studies on enamel hypomineralisation". *European Archives of Paediatric Dentistry* 16.3 (2015): 235-246.

12. Americano Gabriela CA., *et al*. "Relating molar incisor hypomineralization and caries experience using the decayed, missing, or filled index". *Pediatric Dentistry* 38.5 (2016): 419-424.

13. Gurrusquieta., *et al*. "Prevalence of molar incisor hypomineralization in Mexican children". *Journal of Clinical Pediatric Dentistry* 41.1 (2017): 18-21.

14. Saber F., *et al*. "Prevalence of molar incisor hypomineralisation in a group of Egyptian children using the short form: a cross-sectional study". *European Archives of Paediatric Dentistry* 19.5 (2018): 337-345.

15. Dantas-Neta., *et al*. "Factors associated with molar–incisor hypomineralisation in schoolchildren aged 8–10 years: a case–control study". *International Journal of Paediatric Dentistry* 28.6 (2018): 570-577.

16. Koruyucu., *et al*. "Prevalence and etiology of molar-incisor hypomineralization (MIH) in the city of Istanbul". *Journal of Dental Sciences* 13.4 (2018): 318-328.

17. Saitoh Masato., *et al*. "Prevalence of molar incisor hypomineralization and regional differences throughout Japan". *Environmental Health and Preventive Medicine* 23.1 (2018): 55.

18. Samuel Allwyn., *et al*. "Caries status and salivary characteristics of South Indian school children with molar incisor hypomineralization: A cross-sectional study". *Journal of Indian Association of Public Health Dentistry* 15.2 (2017): 135.

19. Rai Archana., *et al*. "Molar Incisor Hypomineralization: Prevalence and Risk Factors Among 7-9 Years Old School Children in Muradnagar, Ghaziabad". *The Open Dentistry Journal* 12 (2018): 714.

20. Jälevik B. "Prevalence and diagnosis of molar-incisor-hypomineralisation (MIH): a systematic review". *European Archives of Paediatric Dentistry* 11.2 (2010): 59-64.

21. Elfrink MEC., *et al*. "Hypomineralized second primary molars: prevalence data in Dutch 5-year-olds". *Caries Research* 42.4 (2008): 282-285.

22. Elfrink MEC., *et al*. "Deciduous molar hypomineralization and molar incisor hypomineralization". *Journal of Dental Research* 91.6 (2012): 551-555.

23. Garot Elsa., *et al*. "Are hypomineralised lesions on second primary molars (HSPM) a predictive sign of molar incisor hypomineralisation (MIH)? A systematic review and a meta-analysis". *Journal of Dentistry* 72 (2018): 8-13.

24. Mittal N and BB Sharma. "Hypomineralised second primary molars: prevalence, defect characteristics and possible association with Molar Incisor Hypomineralisation in Indian children". *European archives of Paediatric Dentistry* 16.6 (2015): 441-447.

25. Negre-Barber A., *et al*. "Hypomineralized second primary molars as predictor of molar incisor hypomineralization". *Scientific reports* 6 (2016): 31929.

26. Owen ML., *et al*. "Hypomineralized second primary molars: prevalence, defect characteristics and relationship with dental caries in Melbourne preschool children". *Australian dental journal* 63.1 (2018): 72-80.

27. Johnsen D., *et al*. "Distribution of enamel defects and the association with respiratory distress in very low birthweight infants". *Journal of Dental Research* 63.1 (1984): 59-64.

28. Aine, L., *et al*. "Enamel defects in primary and permanent teeth of children born prematurely". *Journal of Oral Pathology and Medicine* 29.8 (2000): 403-409.

29. Silva Mihiri J., *et al*. "Etiology of molar incisor hypomineralization–a systematic review". *Community Dentistry and Oral Epidemiology* 44.4 (2016): 342-353.

30. Jälevik Birgitta., *et al*. "Secondary ion mass spectrometry and X-ray microanalysis of hypomineralized enamel in human permanent first molars". *Archives of Oral Biology* 46.3 (2001): 239-247.

31. William., *et al*. "Molar incisor hypomineralization: review and recommendations for clinical management". *Pediatric dentistry* 28.3 (2006): 224-232.

32. Messer LB. "Getting the fluoride balance right: Children in long-term fluoridated communities". *Synopses* 30 (2005): 7-10.

33. Manton David J., *et al*. "Remineralization of enamel subsurface lesions in situ by the use of three commercially available sugar-free gums". *International Journal of Paediatric Dentistry* 18.4 (2008): 284-290.

34. Crombie, Felicity., *et al*. "Resin infiltration of developmentally hypomineralised enamel". *International Journal of Paediatric Dentistry* 24.1 (2014): 51-55.

35. Wright J Timothy. "The etch-bleach-seal technique for managing stained enamel defects in young permanent incisors". *Pediatrics Dentistry* 24.3 (2002): 249-252.

36. Williams JK and AJ Gowans. "Hypomineralised first permanent molars and the orthodontist". *European Journal of Paediatric Dentistry* 4 (2003): 129-132.

Volume 3 Issue 6 June 2019