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Prevalence, aetiology, and treatment of molar incisor hypomineralization in children living in Izmir City (Turkey)

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Abstract

Background: Molar incisor hypomineralization (MIH) is frequent in children and etiology is different.

Aim: The aim of this study was to determine the prevalence, severity, and risk factors of MIH and to investigate the relationship between the severity of the defect in the teeth affected by MIH and the risk factors.

Design: The study included patients at the age range of 9 and 10 years, who were admitted to the clinic of paediatric dentistry in Dokuz Eylul University and had four-first molar teeth (FMT) and eight incisor teeth eruptions in the jaws. The presence of only demarcated opacities in one or several FMT and incisor teeth was defined as MIH1, and the presence of post-eruptive breakdown (PEB), atypical restorations, and extracted teeth was defined as MIH2.

Results: MIH was detected in 142 (11.5%) of 1237 children. Although there was no statistically significant difference between upper and lower FPM and the occurrence of MIH ($P < 0.902$), there was a difference between upper and lower central and lateral teeth ($P < 0.001$; $P < 0.009$). A significant difference was detected between birth week, birthweight, high fever, and asthma/bronchitis and MIH1 and MIH2 ($P < 0.001$).

Conclusion: The prevalence of MIH was found to be 11.5%. It was observed that the patients undergoing low birthweight, preterm delivery, high fever, and asthma/bronchitis were more severely affected by MIH.

KEYWORDS

asthma, birth week, birthweight, high fever, molar incisor hypomineralization

1 | INTRODUCTION

The term molar incisor hypomineralization (MIH) first emerged in 2001 to describe the clinical picture of enamel hypomineralization, in which one or more permanent molars (FMT) are affected and incisors are frequently involved.[1-4] It is stated that many factors play a role in the aetiology of MIH. It is thought that hypomineralization is observed in the maturation stage of the amelogenesis and that ameloblastic activity is systemically damaged in this period.[3,4] In the literature, it is stated that prenatal and post-natal problems, preterm birth, low birthweight, respiratory diseases, high fever, chickenpox, and frequent antibiotic use in early childhood may be effective in the occurrence of MIH.[5-10] MIH can be seen in all or several of FMTs and incisors, and the severity of the defect in the affected teeth may vary. When the number of FMTs affected by MIH increases, the rate of affected incisor teeth also increases. It is seen that incisor teeth are not affected at the same severity and intensity as FMTs. For a patient to be diagnosed with MIH, a total of 12 teeth,

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including FMT and incisors, should be checked for opacity on their surfaces.[11] Clinically, MIH can be seen in colours from white to yellow-brown.[12,13] This is due to the increase in the organic structure of the enamel layer. The enamel of these teeth has more protein content than healthy teeth. Therefore, transmission between acid and hydroxyapatite crystals decreases and the levels of serum albumin, alpha 1 antitrypsin, and type 1 collagen increase. If the colour of the tooth with MIH is brown or yellow, it is stated that these teeth have antithrombin 3 protein.[14] The studies show that the prevalence of MIH varies between 2.8% and 40.2% among the societies.[1,3,5,13,15-22] Although the incidence of MIH was found to be 2.8% in Chinese children,[15] it was 40.2% in Brazilian children.[16] The difference between the studies can result from the index that was used, study method, age range, health systems of the countries, and socio-economic status.[5,11,23-27]

Early diagnosis of the teeth affected by MIH and determining the correct treatment method are very important. In this study, it was aimed to determine the prevalence and severity of MIH in patients admitted to a paediatric dental clinic in a university, to investigate the relationship between MIH severity and birth week, birthweight, asthma/bronchitis, and high fever, and also to plan treatment.

Why this paper is important to paediatric dentists?

- Early recognition of aetiologic factors is important to identify children potentially at risk for developing MIH.
- This study demonstrated that children may present with not only mild forms of MIH but also more severe forms such as enamel breakdown and need for atypical restorations.
- Paediatric dentists should consider that molar and incisor teeth may be more severely affected in low birthweight, premature, high fever, and asthma/bronchitis while they encounter children.

2 | MATERIALS AND METHODS

The study included patients between the ages of 9 and 10 years, who were admitted to Dokuz Eylul University paediatric dental clinic between the dates of 01 January 2015 and 30 December 2018 for the purpose of dental examination. Patients with four FMTs and eight incisor teeth with the eruption of full crown length in the lower-upper jaws were included in the study.

2.1 | Dental examination of the subjects

The examinations were carried out in the clinical setting by drying the teeth under refractor light and using the dental mirror and probe. In the intraoral examination, the European Academy of Paediatric Dentistry (EAPD) criteria were used for MIH.[12] The criteria of the World Health Organization (WHO) for caries assessment (DMFT; D: decay, M: missing, F: filling, T: total) were used in the determination of carious teeth. The teeth with MIH and their severity were recorded, and a form was prepared for each patient to be asked to their mothers. This form included

1. Demographic data:

Age: The age range in this study was 9 and 10 years. Children younger than 9 years, the eruption of lateral teeth is not completed. In the children over 10 years old, because FMT affected by MIH, they are extracted due to caries.

Gender; Male, female

2. Birth data: Patients with birth week of <32 were grouped as preterm (PT) and those with 33-40 weeks as a full term (FT).[28] Patients with the birthweight of <2500 g were considered as low-weighted (LW), and those with the birthweight of 2500-4000 g were considered as normal-weighted (NW).
3. Child's medical history: Parents were asked whether their children had asthma/bronchitis and high fever over 38.5°C for five times or more in a year during the first 3 years of their lives (yes/no).

2.2 | Diagnosis and classification of MIH

Teeth affected by MIH are clinically divided into groups:

1. Demarcated opacity: Change of enamel colour is observed in FMT and/or incisor teeth. This change is in the form of white, yellow, or brown opacities and does not contain caries.
2. Post-eruptive enamel breakdown: In FMT and/or incisor teeth, the colour of the enamel goes up to yellow-brown and loss in the dentine tissue of the tooth and caries is seen.
3. Atypical restoration: Atypical large fillings (amalgam, composite, glass ionomer, crown restorations, and so on), which are not similar to other decay structure in the mouth of the patient, are seen in FMT and/or incisor teeth.
4. Extracted molar due to MIH: Extraction of one or several FMT due to MIH. Demarcated opacity and/or post-eruptive enamel breakdown is seen in other FMT.

TABLE 1 Demographic, birth, and medical history data of patients with MIH

N	MIH (%)
Gender	
Male	73 (51.4)
Female	69 (48.6)
Age	
9	80 (56.3)
10	62 (43.7)
Birth week	
33-40	113 (79.6)
<32	29 (20.4)
Birthweight (g)	
2500-4000	106 (74.6)
2500<	36 (34.4)
Asthma/bronchitis[a]	
Yes	17 (12.0)
No	125 (88.0)
Fever higher than 38.5°C[a]	
Yes	35 (24.6)
No	107 (75.4)

[a]Undergoing asthma/bronchitis and fever higher than 38.5°C for five times and more in the first 3 y of their lives.

2.3 | MIH group

If the patient had only demarcated opacity in one or more of the FMT and incisor teeth, the patient was grouped as MIH1. The patient was grouped as MIH2 in the presence of post-eruptive enamel breakdown, atypical restoration, and extracted molar due to MIH in at least one of the FMT and incisor teeth.

2.4 | Inclusion and exclusion criteria

For the diagnosis of MIH, the condition that at least one of the FMT was affected was sought. In the presence of only the lower-upper affected incisors, they were excluded by considering that the defects in these teeth might be due to other local factors. Patients with systemic disease and syndrome and patients undergoing orthodontic treatment during the examination were excluded from the study. Furthermore, the teeth with MIH lesion smaller than 2 mm were also not included in the study. In order to measure the lesions smaller than 2 mm, periodontal probe was used and the measurements were done according to the mm values on the probe.

2.5 | Treatment

The routine treatment protocol was applied to all patients. The protective fluoride gel (Sultan topical APF 2.0% neutral sodium fluoride) was applied for all 142 patients affected by MIH. For incisor teeth, two teeth with post-eruptive enamel breakdown were performed composite restorations, but no incisor teeth were extracted. Composite (3M Espe Filtek Z250), glass ionomer (Ketac Molar Easymix), and amalgam (Cavex Non Gamma-2 capsule) restorations were performed in 191 of 237 FMT with post-eruptive enamel breakdown, and 11 FMT with excessive crown damage were extracted. The patients were called for a regular follow-up.

2.6 | Ethical approval

For research ethics, approval was received from the noninvasive ethics committee of the Medical Faculty of DEU (No: 2018/28-07). Moreover, written informed consent was obtained from parents.

2.7 | Statistical analysis

Statistical package for the social sciences (SPSS) 15.0 manufactured by IBM was used for the statistical analysis. Statistical analyses of the differences between the groups were performed by using the chi-square test, and nonparametric analysis was used to determine the relationships between variables. In all analyses, the value of $P < 0.05$ was accepted as statistically significant.

3 | RESULTS

MIH was detected in 142 (11.5%) of 1237 patients who underwent intraoral examinations. There was no statistically significant difference between gender and MIH ($P = 0.958$). The distributions of patients with MIH according to gender, age, birth week, birthweight, asthma/bronchitis, and high fever over 38.5°C in the first 3 years after delivery are presented (Table 1).

It was observed that 644 (37.8%) of 1704 (FMT and incisors) teeth of 142 patients were affected by MIH (mean 2.79 ± 1.0). In this study, most of the teeth affected by MIH are FMT. MIH affected FMT (n = 493, 76.6%) more frequently than incisors (n = 151, 23.4%; $P = 0.001$). The least affected teeth were mandibular second incisors. The prevalence of MIH did not differ significantly between the maxillary and mandibular FMT ($P = 0.902$), but a significant difference was found between the maxillary and mandibular central and lateral incisors ($P = 0.001$ and $P = 0.009$). Among incisors, upper central incisors (n = 107 16.6%) were most commonly affected. Maxillary central and lateral incisors were found to be more affected by MIH compared to mandibular central and lateral incisors. Although this rate was 20.2% in maxillary and mandibular central incisors, it

TABLE 2 Distribution of permanent teeth affected by molar incisor hypomineralization

N (%)	MIH Maxillary arch	MIH Mandibular arch	*P* value	MIH Total number
First Molars	253 (39.3)	240 (37.3)	0.902	493 (76.6)
Central incisor	107 (16.6)	23 (3.6)	0.001[a]	130 (20.2)
Lateral incisor	19 (2.9)	2 (0.3)	0.009[b]	21 (3.2)
Total	379 (58.9)	265 (41.1)		644 (100.0)

[a]Chi-square test.
[b]Fisher's exact test.

was 3.2% in maxillary and mandibular lateral incisors teeth (Table 2).

It was found that the number of teeth with demarcated opacity ($P = 0.001$) was higher in the maxillary FMT, the number of teeth with post-eruptive enamel breakdown ($P = 0.023$) and atypical restoration ($P = 0.003$) was higher in the mandibular FMT, and the difference was statistically significant. No extracted and filled teeth due to MIH were encountered in the incisor teeth (Table 3).

Maxillary FMT and incisor teeth (n = 379) were detected to be affected by MIH more than the mandibular teeth (n = 265) ($P < 0.001$). In addition, the number of atypical restorations was higher in the mandibular FMT ($P = 0.004$), but there was no difference in the number of extracted molar teeth due to MIH (Table 4).

There was a statistically significant difference between birth week, birthweight, health problems during the first 3 years of life (asthma/bronchitis and fever), and MIH1 and MIH2 ($P < 0.001$) (Table 5).

Of 236 FMT with demarcated opacity and post-eruptive enamel breakdown, 191 (80.9%) were filled, 11 (3.4%) were extracted, and 34 (14.4%) were controlled. FMTs with post-eruptive enamel breakdown were performed more fillings compared to those with demarcated opacity (Table 6).

4 | DISCUSSION

Early detection of teeth affected by MIH is very important in childhood. The patients can have complaints of widespread caries and pain, which results in enamel loss at an early age. In our study, the prevalence of MIH was found to be 11.5% in children between the aged of 9 and 10 years. Although the rate of MIH was the same as ours in the study of Wuollet et al.,[24] it was very close to our findings in the studies of Muratbegovic et al.[1] (12.3%) and Parikh et al.[25] (9.7%). In the study of Koruyucu et al.,[27] which was conducted on children at the age of 8 and 11 years in Istanbul, the rate of MIH was detected to be 9.9% in the age group of 8 years and 18.2% in the age group of 11 years and it was stated that it increased with age. Although the prevalence of MIH was reported to be higher than ours by some researchers,[1,3,16-21,29] Preusser et al.[5] (5.9%) and Cho et al.[15] (2.8%) found a lower rate. Weerheijm et al.[12] stated that this difference between the data might be caused by differences in the examination conditions. The selection of older children in the study group, the inclusion of lesions smaller than 2 mm in the study, and drying of teeth during clinical examination and clinical study according to the school are suggested to lead to a higher prevalence of MIH.[9,10,16,18,30] In the meta-analysis performed by Schwendicke et al,[31] the mean prevalence of MIH was found to be 13.1% and they detected differences among the countries. We also suggest that one reason for differences in data among the studies is due to examination conditions. Another reason is that the occurrence rate of MIH can differ when children undergoing preterm birth, low birthweight, frequent high fever, frequent respiratory diseases, and associated use of antibiotics are included in the study population.

Therefore, in this study, we examined birth data and the child's medical history in addition to giving more detailed information about the level and incidence of MIH, which is the strength of our study. As in many studies,[15,20,27] there was no relationship between gender and MIH in our study. Zawaideh et al,[3] however, reported the rate of MIH to be higher in girls and Jalevik et al[20] to be higher in boys.

The age range in this study was 9-10 years. This is because FMT and incisors have fully erupted in the mouth in this age group. In studies performed on children younger than 9 years, the eruption of lateral teeth is not complete. In the children over 10 years old, because FMT affected by MIH are more susceptible to caries, they are extracted. Both conditions can cause MIH to be reported incompletely.[9]

The incidence of MIH in maxillary and mandibular FMT was very close to each other. Similarly, many researchers have found no difference between maxillary and mandibular FMT and the incidence of MIH,[1,12,15,18,20] whereas Jasulaityte et al[29] and Subramaniam et al[33] reported MIH to be seen more commonly in the lower jaw and Preusser et al[5] reported that significantly more maxillary molars were affected.

In this study, it was observed that although FMT (76.6%) were more affected by MIH, incisors were less affected (23.4%). Similarly, many researchers stated that FMT were more affected compared to incisor teeth[8,10,12,15,16,20].

TABLE 3 Distribution of teeth affected by MIH according to teeth and defect scores

N (%)	Maxillary Molar (16 + 26)	Mandibular Molar (36 + 46)	*P*-value	Maxillary Central (11 + 21)	Mandibular Central (31 + 41)	*P*-value	Maxillary Lateral (12 + 22)	Mandibular Lateral (32 + 42)	*P*-value
Demarcated opacity	164 (25.5)	90 (1.9)	0.001*	107 (16.6)	23 (3.6)	0.001*	17 (2.6)	2 (0.3)	0.001*
PEB	77 (12.0)	97 (15.1)	0.023*	-	-		2 (0.3)	-	-
Atypical restoration	6 (0.9)	22 (3.4)	0.003*	-	-		-	-	-
Extracted molar due to MIH	6 (0.9)	6 (0.9)	1.000	-	-		-	-	-
MIH	253 (39.3)	240 (37.3)		107 (16.6)	23 (3.6)		19 (2.9)	2 (0.3)	
No defect	31	44		177	261		267	282	

Abbreviations: MIH, molar incisor hypomineralization; PEB, post-eruptive enamel breakdown.
Chi-square test *$P < 0.05$.

Jasulaityte et al,[29] however, found no statistically significant correlation between the number of molars and the number of incisors that were affected.

As many researchers, we classified MIH in four categories (demarcated opacities, PEB, atypical restorations, and extracted FPMs, filling, extracted molar due to MIH).[10,27,35] Our aim was to present the severity and incidence of MIH in more detail.

In the incisors teeth, the rate of being affected by MIH is the highest in the maxillary centrals (16.3%) and the lowest in the mandibular laterals (0.3%). We observed more demarcated opacities lesions in FMT and incisors. In the same way, researchers reported the lesions of demarcated opacities to be more frequent.[3,11,17,29,34,36] We think that especially FMT can change towards PEB and atypical restorations in the long term if necessary preventive treatments are not applied to young patients with demarcated opacities.

In this study, no difference was detected in the incidence of MIH between maxillary and mandibular FMT. When we divided the teeth into groups according to their lesions, however, it was observed that the numbers of PEB and atypical restoration were higher in mandibular FMT. We attributed this to the earlier eruption of mandibular FMT and rapid change of existing demarcated opacities to PEB. In the study of Subramaniam et al,[33] they stated a similar view to ours on this issue.

In the recent years, the studies on the aetiology of MIH have shown that the incidence of MIH is higher in children undergoing high fever and respiratory diseases in the first years after birth, preterm delivery, and low birthweight.[8,17,28,37,38] The maturation stage of tooth enamel starts in the last trimester of pregnancy and continues up to first 3 years after birth. Due to premature birth, tooth enamel cannot complete the development of maturation and enamel defects and MIH can develop in patients. Similarly, in the first years of life, the development of health problems such as asthma, frequent upper respiratory infections (adenoid infections), and high fever affects the ameloblastic activity during enamel mineralization and enamel defects are seen in FMT.

In this study, 20.4% of the cases with MIH were preterm, 25.4% of them had low birthweight, and all of these patients were those included in the MIH2 group. Brogardh-Roth et al[28] reported that the incidence of MIH was higher in preterm children (38%) than in controls (16%) and low birthweight increased the risk of MIH. Moreover, they stated that severe caries problems were more frequently encountered in preterm children. In the study of Sönmez et al,[38] a relationship between premature and low birthweighted patients and MIH was shown. Similar to the studies of these two researchers,[36,38] we observed PEB, atypical restoration, and extracted molar due to MIH in patients with preterm birth and low birthweight. There are also reports presenting different results, however; Koruyucu et al[27]

TABLE 4 Distribution of teeth affected by MIH according to the jaws and defect scores

First Molars and Incisors N (%)	Maxillary	Mandibular	*P*-value	Total number
Demarcated opacity	288 (34.7)	140 (21.7)	0.001*	428 (56.4)
Post-eruptive enamel breakdown	79 (12.4)	97 (15.1)	0.176	176 (27.5)
Atypical restoration	6 (0.9)	22 (3.4)	0.004*	28 (4.3)
Extracted molar due to MIH	6 (0.9)	6 (0.9)	1.000	12 (1.8)
MIH	379 (58.9)	265 (41.1)	0.001*	644 (100.0)
No defect	473	433		724
Total	852	852		1704

Chi-square test *$P < 0.05$.

TABLE 5 Distribution of MIH1 and MIH2 patients according to birth week, birthweight, and health problems during the first 3 y of their lives

Variables	MIH1 n (%)	MIH2 n (%)	*P*-value
Birth week			
33-40	60 (42.3)	53 (37.3)	<0.001
<32	-	29 (20.4)	
Birthweight (g)			
2500-4000	60 (42.3)	46 (32.4)	<0.001
>2500	-	36 (25.4)	
Asthma/bronchitis (year/5 and more)			
No	60 (42.3)	65 (45.8)	<0.001
Yes	-	17 (11.9)	
Fever (year/5 and more)			
No	55 (38.7)	52 (36.7)	<0.001
Yes	5 (3.5)	30 (21.1)	

Note: MIH1: The presence of only demarcated opacity in one or several of the FMT and incisor teeth; MIH2: The presence of post-eruptive enamel breakdown, atypical restoration, and extracted molar due to MIH in at least one of the FMT and incisor teeth.
Chi-square test $P < 0.05$.

and Allazzam et al[35] reported no relationship between low birthweight and MIH.

In this study, it was observed that patients undergoing asthma/bronchitis and high fever were more in the MIH2 group. Patients receiving asthma/bronchitis treatment in early childhood have been reported to have impaired matrix protein structure due to lack of oxygen and to develop MIH.[27,35] Similarly, frequently developing high fever in the formation process of the enamel causes degeneration in the enamel prisms, which is associated with MIH development.[38] When these results are considered, patients with severe MIH should be questioned in terms of whether they have had premature birth, low birthweight, asthma/bronchitis, and high fever in early childhood.

Studies indicate that the teeth affected by MIH are prone to dental caries at a twofold to 10-fold higher rate. In addition, preventive and protective treatment with early diagnosis has been reported to be very important due to the rapid breakdown of the enamel structure and the rapid development of the symptoms.[20,36,39-41] Protective therapies are recommended in the absence of any substance loss in tooth, and conservative or invasive restorations are recommended in cases with substance loss.[40,41] In our study, protective treatment (Topical fluorine) was applied in all patients affected by MIH and restoration and/or extraction was performed in demarcated opacities and PEB teeth.

This study has several limitations. The most important ones of these limitations are those: it was conducted in a single centre, a sociocultural evaluation was not performed, and oral hygiene behaviours were not evaluated. It should also be kept in mind that there may be recall bias in the investigation of asthma/bronchitis and fever. On the other hand, the high

TABLE 6 Treatments applied in cases of MIH

	MIH Treatment Demarcated opacity		MIH Treatment PEB		
Molars teeth	Maxillary	Mandibular	Maxillary	Mandibular	Total
Filling (Composite, amalgam, glass ionomer)	8 (3.4)	20 (8.5)	74 (31.3)	89 (37.7)	191 (80.9)
Missing	-	-	3 (1.3)	8 (3.4)	11 (4.7)
Followed up	25 (10.6)	7 (3.0)	2 (0.8)	-	34 (14.4)

Abbreviation: PEB, Post-erupted breakdown.

number of our patient population and evaluation with a detailed examination can be considered as the strengths of our study.

5 | CONCLUSION

In this study, the prevalence of MIH was found to be 11.5% in patients between the ages of 9 and 10 years. The most commonly observed lesion was creamy white opacity. It, however, was observed that patients with low birthweight, preterm delivery, high fever, and asthma/bronchitis were more severely affected by MIH, and there was widespread caries especially in FMT. The immediate dental treatment and regular follow-up of the patients diagnosed with MIH are very important.

AUTHORS' CONTRIBUTION

G.K. and M.Ç. conceived the ideas; G.K. and B.K. collected the data; G.K. and H.E. analysed the data; G.K., M.Ç., and B.K. contributed to conception and design; G.K. drafted manuscript; All authors have made substantive contribution to this study and/or manuscript, and all have reviewed the final paper prior to its submission.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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REFERENCES

1. Muratbegovic A, Markovic N, Selimovic MG. Molar incisor hypomineralisation in Bosnia and Herzegovina: prevalence, aetiology and clinical consequences in medium caries activity population. *Eur Arch Paediatr Dent*. 2007;8:189-194.
2. Mast P, Rodrigueztapia MT, Daeniker L, Krejci I. Understanding MIH: definition, epidemiology, differential diagnosis and new treatment guidelines. *Eur J Paediatr Dent*. 2013;14:204-208.
3. Zawaideh FI, Al-Jundi SH, Al-Jaljoli MH. Molar incisor hypomineralisation: prevalence in Jordanian children and clinical characteristics. *Eur Arch Paediatr Dent*. 2011;12:31-36.
4. Weerheijm KL, Elfrink ME, Kilpatrick N. Molar incisor hypomineralization and hypomineralized second primary molars: diagnosis, prevalence, and etiology. In: Drummond BK, Kilpatrick N, eds. *Planning and Care for Children and Adolescents with Dental Enamel Defects*. Heidelberg: Springer; 2015:31-44.
5. Preusser SE, Ferring V, Wleklinski C, Wetzel WE. Prevalence and severity of molar incisor hypomineralization in a region of Germany – a brief communication. *J Public Health Dent*. 2007;67:148-150.
6. Whatling R, Fearne JM. Molar incisor hypomineralization: a study of aetiological factors in a group of UK children. *Int J Paediatr Dent*. 2008;18:155-162.
7. Laisi S, Ess A, Sahlberg C, Arvio P, Lukinmaa PL, Alaluusua S. Amoxicillin may cause molar incisor hypomineralization. *J Dent Res*. 2009;88:132-136.
8. Arrow P. Risk factors in the occurrence of enamel defects of the first permanent molars among schoolchildren in Western Australia. *Community Dent Oral Epidemiol*. 2009;37:405-415.
9. Crombie F, Manton D, Kilpatrick N. Aetiology of molar-incisor hypomineralization: a critical review. *Int J Paediatr Dent*. 2009;19:73-83.
10. da Costa-Silva CM, Ambrosano GM, Jeremias F, De Souza JF, Mialhe FL. Increase in severity of molar–incisor hypomineralization and its relationship with the colour of enamel opacity: a prospective cohort study. *Int J Paediatr Dent*. 2011;21:333-341.
11. Jalevik B, Noren JG. Enamel hypomineralization of permanent first molars: a morphological study and survey of possible aetiological factors. *Int J Paediatr Dent*. 2000;10:278-289.
12. Weerheijm KL, Duggal M, Mejare I, et 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;4:110-113.
13. Wuollet E, Laisi S, Salmela E, Ess A, Alaluusua S. Molar–incisor hypomineralization and the association with childhood illnesses and antibiotics in a group of Finnish children. *Acta Odontol Scand*. 2016;74:416-422.
14. dos Santos MPA, Maia LC. Molar incisor hypomineralization: morphological, aetiological, epidemiological and clinical considerations. In: Li DM-Y, eds. *Contemporary Approach to Dental Caries*. London, UK: InTechopen; 2012:423-446.
15. Cho SY, Ki Y, Chu V. Molar incisor hypomineralization in Hong Kong Chinese children. *Int J Paediatr Dent*. 2008;18:348-352.
16. Soviero V, Haubek D, Trindade C, Da Matta T, Poulsen S. Prevalence and distribution of demarcated opacities and their sequelae in permanent 1st molars and incisors in 7 to 13 year old Brazilian children. *Acta Odontol Scand*. 2009;67:170-175.
17. Ghanim A, Morgan M, Mariño R, Bailey D, Manton D. Molar -incisor hypomineralisation: prevalence and defect characteristics in Iraqi children. *Int J Paediatr Dent*. 2011;21:413-421.
18. Calderara PC, Gerthoux PM, Mocarelli P, Lukinmaa PL, Tramacere PL, Alaluusua S. The prevalence of molar incisor hypomineralization (MIH) in a group of Italian school children. *Eur J Paediatr Dent*. 2005;6:79-83.
19. Zagdwon AM, Toumba KJ, Curzon MEJ. The prevalence of developmental enamel defects in permanent molars in a group of English school children. *Eur J Paediatr Dent*. 2002;3:91-96.
20. Jalevik B, Klingberg GA, Barrega°rd L, Nore′n JG. The prevalence of demarcated opacities in permanent 1st molars in a group of Swedish children. *Acta Odontol Scand*. 2001;59:255-260.
21. Garcia-Margarit M, Catala-Pizarro M, Montiel-Company JM, Almerich-Silla JM. Epidemiologic study of molar-incisor hypomineralization in 8-year-old Spanish children. *Int J Paediatr Dent*. 2014;24:14-22.
22. Ghanim AM, Bagheri R, Golkari A, Manton D. Molar-incisor hypomineralisation: a prevalence study amongst primary schoolchildren of Shiraz, Iran. *Eur Arch Paediatr Dent*. 2014;15:75-82.
23. Durmuş B, Abbasoğlu Z, Kargul B. Possible medical etiological factors and characteristics of molar incisor hypomineralization in a group of Turkish children. *Acta Stomatol Croat*. 2013;47:297-305.

24. Wuollet E, Laisi S, Salmela E, Ess A, Alaluusua S. Background factors of molar-incisor hypomineralization in a group of Finnish children. *Acta Odontol Scand*. 2014;72:963-969.
25. Parikh DR, Ganesh M, Bhaskar V. Prevalence and characteristics of molar incisor hypomineralization (MIH) in the child population residing in Gandhinagar, Gujarat, India. *Eur Arch Paediatr Dent*. 2012;13:21-26.
26. Dietrich G, Sperling S, Hetzer G. Molar incisor hypomineralisation in a group of children and adolescents living in Dresden (Germany). *Eur J Paediatr Dent*. 2003;4:133-137.
27. Koruyucu M, Özel S, Tuna EB. Prevalence and etiology of molar-incisor hypomineralization (MIH) in the city of Istanbul. *J Dent Sci*. 2018;13:318-328.
28. Brogardh-Roth S, Matsson L, Klingberg G. Molar-incisor hypomineralization and oral hygiene in 10- to- 12-yr-old Swedish children born preterm. *Eur J Oral Sci*. 2011;119:33-39.
29. Jasulaityte L, Veerkamp JS, Weerheijm KL. Molar incisor hypomineralization: review and prevalence data from the study of primary school children in Kaunas/Lithuania. *Eur Arch Paediatr Dent*. 2007;8:87-94.
30. Lygidakis NA, Dimou G, Briseniou E. Molar-incisor-hypomineralisation (MIH). Retrospective clinical study in Greek children. I. Prevalence and defect characteristics. *Eur Arch Paediatr Dent*. 2008;9:200-206.
31. Schwendickea F, Elhennawya K, Redaa S, Bekesb K, Mantonc DJ, Krois J. Global burden of molar incisor hypomineralization. *J Dent*. 2018;68:10-18.
32. Ng JJ, Eu OC, Nair R, Hong CH. Prevalence of molar incisor hypomineralization (MIH) in Singaporean children. *Int J Paediatr Dent*. 2015;25:73-78.
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;7:11-15.
34. Wogelius P, Haubek D, Poulsen S. Prevalence and distribution of demarcated opacities in permanent 1st molars and incisors in 6 to 8 -years -old Danish children. *Acta Odontol Scand*. 2008;66:58-64.
35. Allazzam SM, Alaki SM, El Meligy OAS. Molar incisor hypomineralization, prevalence, and etiology. *Int J Dent*. 2014;2014:234508.
36. Mittal NP, Goyal A, Gauba K. Molar incisor hypomineralisation: prevalence and clinical presentation in school children of the northern region of India. *Eur Arch Paediatr Dent*. 2014;15:11-18.
37. Americano GC, Jacobsen PE, Soviero VM, Haubek D. A systematic review on the association between molar incisor hypomineralization and dental caries. *Int J Paediatr Dent*. 2017;27:11-21.
38. Sonmez H, Yıldırım G, Bezgin T. Putative factors associated with molar incisor hypomineralisation: an epidemiological study. *Eur Arch Paediatr Dent*. 2013;14:375-380.
39. Leppaniemi A, Lukinmaa PL, Alaluusua S. Nonfluoride hypomineralizations in the permanent first molars and their impact on the treatment need. *Caries Res*. 2001;35:36-40.
40. Lygidakis NA, Wong F, Jälevik B, Vierrou AM, Alaluusua S, Espelid I. Best clinical practice guidance for clinicians dealing with children presenting with molar-incisor hypomineralisation(MIH): an EAPD policy document. *Eur Arch Paediatr Dent*. 2010;11:75-81.
41. Fragelli CM, Souza JF, Jeremias F, Cordeiro Rde C, Santos-Pinto L. Molar incisor hypomineralization (MIH): conservative treatment management to restore affected teeth. *Braz Oral Res*. 2015;29:1-7.

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