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Original Article (Pages: 14341-14347)

Prevalence of Molar Incisor Hypomineralisation among School Children of Sari, Iran

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Abstract

Background: The purpose of this study was to estimate the prevalence of molar incisor Hypomineralisation (MIH) in a group of school children aged 7 to 12 years, in sari.

Materials and Methods: 700 school children (350 males and 350 females) were randomly selected for participation in this descriptive cross-sectional study. The prevalence of MIH was, initially, determined through clinical examination. The participants with MIH were also interviewed, and Related Data was recorded in questionnaires filled by the examiner. The data of study was analyzed using the Chi-square, Fisher's exact, and Mann-Whitney U tests through SPSS version 17. P-vale less than 0.05 was considered statistically significant.

Results: The prevalence of MIH was 20.2 % (n =142). Prevalence rate calculated in the studied group was 21.1% (males=74) and 19.4% (females=68).There was no significant difference between the two genders in terms of the prevalence and intensity of the MIH (P=0.4).

Conclusion: The results revealed that MIH is of moderate prevalence in Sari primary schools, and its prevalence is not significantly associated with gender.

Key Words: Hypomineralization, Incisors, Molar, Prevalence.

*Please cite this article as: Hali H, Molania Jelodar T, Madian M, Gohardehi Sh, Moosazadeh M, Salehi M. Prevalence of Molar Incisor Hypomineralisation among School Children of Sari, Iran. Int J Pediatr 2021; 9(9): 14341-14347. DOI: **10.22038/ijp.2020.51135.4068**

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Received date: Aug. 14, 2020; Accepted date: Aug. 22, 2020

1- INTRODUCTION

Any disturbance in the evolution of the enamel leads to complications in the microscopic structure and appearance of the enamel. Enamel defect is one of the developmental abnormalities that may be observed alone or in combination with other tissue dysplasias of the body (1). Ameloblast cells are very sensitive to oxygen deprivation and can significantly affect the occurrence of lesion. It is also possible that hypomineralization is associated with some disorders and systemic diseases, as well as environmental and genetic factors. Following any qualitative and quantitative defects in the structure of enamel and dentin, teeth are highly susceptible to decay and are at risk of loss (1). Hypomineralization of molars and incisors refers to enamel lesions of systemic origin that involve one or more molar teeth, usually the first permanent molars, and may or may not be associated with enamel lesions in permanent incisor teeth (2).

In MIH, enamel defects in permanent molars and incisors can occur asymmetrically, whereas in a hypoplasia the defects are usually symmetrical. Here again the question arises as to whether there is an environmental factor for MIH or that it may be caused by a genetic factor. Although, this asymmetry can be explained by different active and silent groups of amlo blasts, threshold levels are not known for the etiological factors of enamel defects. This is especially related to the fact that ameloblasts are more or less sensitive at different stages of enamel formation. Similarly, there may be chronic diseases without systemic symptoms that are severe enough for the child to develop enamel apoptosis, for example, an undetectable viral infection that can lead to enamel opacities. Limited information about MIH is currently available. In different studies, the prevalence of MIH has been reported in the range of 3.25-6 (3).

One of the most common manifestations of MIH in routine clinical examination in many teeth, and often in permanent first molar teeth, is the opacities on the enamel. The degree of tooth involvement varies from one point at the apex to complete crown involvement. In the Netherlands, due to the similarity of color and consistency of these lesions to Dutch cheese, molars with idiopathic enamel disorder have been called chees molar (2, 1). Among the factors that cause MIH are allergic, chemical, infectious, metabolic, neurological, nutritional, syndromic, antibiotic use (especially amoxicillin) and factors related to time before, during and after birth. (1). Breast milk due to its PCBs (polychlorinated biphenyls) and dioxin is an important risk factor in studies on MIH (2).

As stated above, there might be a relationship between dioxin intake through breast milk after prolonged breastfeeding and developmental defects of the baby's teeth. Furthermore, due to the high sensitivity of ameloblasts to oxygen supply, they might be influenced by respiratory disorders associated with hypoxia during labor, respiratory diseases such as asthma and bronchitis, and pneumonia (4). Children, who usually take antibiotics, are those usually sick; so, it is not possible to determine whether the drug or disease is the cause of the defect. Tetracycline is the only antibiotic that causes developmental defects in tooth tissues (5).

The use of amoxicillin at an early age can also be effective in causing MIH (6). There are few published studies on MIH in our country. Due to the lack of comprehensive information on these deficiencies in Mazandaran province, the purpose of this study was to investigate the prevalence of Müller-Sanaia hypomineralization in children in Sari.

2- MATERIALS AND METHODS

This study is a descriptive cross-sectional study and its sampling was done by random selection method. 700 children aged 7-12 years were selected from primary schools in Sari according in accordance with the study of Ahmadi et al. (7).

In this study, the city of Sari was divided into four regions based on the population. In each region, public and non-profit primary schools for boys and girls were numbered, and in order to eliminate the socio-economic status as an intervening factor, with the help of a random table of numbers in each region, a public school for boys, a public school for girls, a non-profit school for boys and a non-profit school for girls were selected. Necessary coordination was done with the education organization of Mazandaran province; and finally, with the education departments of districts 1 and 2 of Sari city to enter schools and examines students. Eventually, in each school, the completion of the questionnaires initiated after the necessary coordinations with school officials and health educators (8).

The questionnaire included clinical examinations performed by a previously calibrated and trained examiner using dental examination equipment including disposable dental bed mirror, dental gauze and direct disposable catheter. The diagnosis of MIH was based on the previously explained criteria as well as the cases described in the pediatric ward by the supervisor. In this study, which was conducted to investigate the prevalence of MIH in children aged 7-12 years in Sari, 700 children were examined. They comprised of 350 (50%) girls and 350 (50%) boys, who had complete permanent upper and lower first molar teeth.

Data analysis was performed on SPSS software version 17, using chi-square (square-Chi) and t-tests.

3- RESULTS

In this study, which was conducted to investigate the prevalence of MIH in children aged 7-12 years in Sari, 700 children including 350 girls (50%) and 350 boys (50%) were studied. The prevalence of MIH was 20.2% (142 cases) among the participants, which was 21.1% in boys (74 cases) and 19.4% (68 cases) in girls. Chi-square test did not show a significant relationship between gender and the prevalence of MIH ($p = 0.4$) (**Table 1**).

The frequency and prevalence of MIH of high permanent first molars and low permanent first molars were 8.4% ($n = 59$) and 8.1% ($n = 57$), respectively. Thus, the prevalence of MIH in both types of teeth is almost the same. (**Table 2**)

The prevalence of MIH of high incisor was 5.1% ($n = 36$) and low incisor was 2.3% ($n = 16$). The frequency of MIH in high incisor teeth was higher than that of low incisors. (**Table 3**)

The results of the present study revealed that the upper and lower incisors and the first permanent upper and lower first molars are more of white-cream type. Most diffuse opsites observed in incisors were diffuse-line type and the first permanent dermulars were diffuse-patchy. Regarding hypoplasia, the most common condition observed in affected teeth in the present study was pitted type. In terms of the extent of involvement of the upper and lower incisor teeth and the lower permanent first molar, the most involvement was less than 1.3 of the tooth surface, and in the upper permanent first molar teeth, between 2 and 2 tooth surfaces were observed (**Tables 4-7**).

Table-1: Distribution Frequency of MIH among the participants in terms of Gender

MIH / gender	having		not having		Total	
	Frequency	Percentage	Frequency	Percentage	Frequency	Percentage
Boy	74	21.1	276	87.9	350	100
Girl	68	19.4	282	80.6	350	100
Total	142	20.2	558	79.7	700	100
Test	p = 0.4					

Table-2: Frequency distribution and prevalence of MIH by type of tooth involved (high permanent first molars, low permanent first molars)

Patients with MIH / Tooth type	Number	Prevalence (Percentage)
high permanent first molars	59	8.4
low permanent first molars	57	8.1

Table-3: Frequency distribution of patients with MIH by tooth type (high incisor and low incisor)

Patients with MIH Tooth type	Number	Prevalence (Percentage)
high incisor	36	5.1
low incisor	16	2.3

Table-4: Distribution of involvement of high incisor teeth with MIH by tables A, B, C, D (DDE table)

Table	Condition	Frequency	Percentage
A (Color)	Normal	0	0
	White-cream	34	94.5
	Yellow- brown	2	5.5
B (Diffuse opacities)	Normal	0	0
	Diffuse line	34	94.5
	Diffuse-patchy	2	5.5
	Diffuse-confluent	0	0
	Confluent / patchi + staining + loss of enamel	0	0
C (Hypoplasia)	Normal	4	11.1
	Pits	26	72.2
	Missing enamel	3	8.3
	Any other defects	3	8.3
D (Size)	Normal	0	0
	< 1.3	34	94.5
	1.3 << 2.3	2	5.5
	At least > 2.3	0	0
Total	36		

Table-5: Distribution of involvement of lower incisor teeth with MIH by tables A, B, C, D (DDE table)

Table	Condition	Frequency	Percentage
A (Color)	Normal	0	0
	White-cream	16	100
	Yellow- brown	0	0
B (Diffuse opacities)	Normal	0	0
	Diffuse-line	16	100
	Diffuse-patch	0	0
	Diffuse-confluent	0	0
	Confluent/patchi +staining+loss of enamel	0	0
C (Hypoplasia)	Normal	4	25
	Pits	12	75
	Missing enamel	0	0
	Any other defects	0	0
D (Size)	Normal	0	0
	< 1.3	16	100
	1.3 << 2.3	0	0
	At least > 2.3	0	0
Total	16		

Table-6: Distribution of involvement of high permanent first molars with MIH by tables A, B, C, D (DDE Table)

Table	Condition	Frequency	Percentage
A (Color)	Normal	0	0
	White-cream	47	79.67
	Yellow- brown	12	20.33
B (Diffuse opacities)	Normal	0	0
	Diffuse-line	22	37.28
	Diffuse-patchy	28	47.46
	Diffuse-confluen	99	15.26
	Confluent/patchi +staining+loss of enamel	0	0
C (Hypoplasia)	Normal	10	16.95
	Pits	8	13.56
	Missing enamel	4	6.78
	Any other defects	37	62.71
D (Size)	Normal	0	0
	< 1.3	12	20.33
	1.3 << 2.3	47	79.67
	At least > 2.3	0	0
Total	59		

Table-7: Distribution of involvement of lower permanent first molars with MIH by tables A, B, C, D (DDE table)

Table	Condition	Frequency	Percentage
A (Color)	Normal	0	0
	White-cream	50	87.72
	Yellow- brown	7	12.28
B (Diffuse opacities)	Normal	0	0
	Diffuse-line	28	49.13
	Diffuse-patchy	29	50.87
	Diffuse-confluent	0	0
	Confluent/patchi +staining+loss of enamel	0	0
C (Hypoplasia)	Normal	5	8.77
	Pits	7	12.28
	Missing enamel	5	8.77
	Any other defects	40	70.18
D (Size)	Normal	0	0
	< 1.3	30	52.64
	1.3 << 2.3	27	47.36
	At least > 2.3	0	0
Total	57		

4- DISCUSSION

In this study, which was performed to investigate the prevalence of MIH in children aged 7-12 years in Sari, 700 children, including 350 (50%) girls and 350 (50%) boys, were studied. The prevalence of MIH among the participants was 20.2% (142 people). Mahmoudian et al., in 2000, throgh examining children aged 7-12 years in Isfahan, showed that the prevalence of enamel defects in boys is 26.7% and in girls is 27.4%. However, no significant relationship was observed between gender and the prevalence of enamel defects (9). In 2003, Mehran et al., conducted a study on children aged 8-9 in Tehran. In that study, the prevalence of enamel defects was 32.2%, 30.1% in boys and 34.2% in girls (10). Leppaniemi et al., in 2001 in the Netherlands reported the prevalence of enamel defects in the first permanent molars of children aged 7-13 years, 19.3% with a higher prevalence in the maxilla (11).

Few studies have been conducted on the prevalence of enamel defects and each has examined a specific type of defects. In 1993, Mousavi et al., reported a prevalence of hypoplasia following trauma to the anterior teeth of children aged 8-12 years in Tehran (15.4%) (12). Weerheijm in 2003, studied on 497 children in the Neatherlands. He found that 142 (14.3%) teeth out of 989 Permanent molars had different degrees of enamel defects (4). Tabari in 1372-73 reported the prevalence of hypoplasia of the first permanent molars, as 14% and 17.5%, respectively, in the north and center of Tehran at the age of 13-18 years. The statistical difference observed in this study as compared to the previous ones can be due to the wide age range included in the study and its statistical population (13). Rugg-Gunn et al., (1998) examined enamel developmental defects in children 6-2 years of age. They have reported a positive correlation between the four factors of low birth weight, drinking water in low volume, drinking milk by Glasses from the

age of 12 months, and living in rural areas (14). The presence of enamel defects in children makes them highly susceptible to caries and complex treatments at an early age. According to Koch et al., the etiology of enamel hypomineralization is divided into three groups: genetic, acquired and idiopathic factors (15).

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