

Molar incisor hypomineralization: Analysis of asymmetry of lesions

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ABSTRACT

Clinically, Molar-Incisor Hypomineralization (MIH) lesions are not distributed symmetrically, and their severity varies even within the same arcade. Aim: To estimate the frequency of asymmetries in hypomineralized lesions on permanent molars and incisors of children with MIH. Methods: Three pediatric dentists, calibrated following the diagnostic criteria of Mathu-Muju and Wright (2006) (Kappa 0.87) identified presence and severity of opacities on molars and incisors of patients with MIH. Six pairs of teeth (permanent maxillary and mandibular first molars, central and lateral incisors) were evaluated in each patient. Degree of lesion severity (0–none, 1–mild, 2–moderate, 3–severe) was recorded for each tooth. For each pair containing any affected teeth, asymmetry of presence (one tooth in the pair with lesion and the other intact), asymmetry of severity (both teeth with lesions but with different degrees of severity) or symmetry of severity (both affected teeth with the same degree of severity) were evaluated. The recorded values were entered into

a database to calculate percentages, 95% confidence intervals and Chi-Square test for comparisons. Results: The sample consisted of 475 of the 1032 pairs of teeth evaluated in the 172 patients included in the study, mean age 11 ± 2.2 years, and 50% female. Asymmetry was found for 67.5% (63.1 - 71.7) of the pairs of the studied teeth. There was a significant relationship between asymmetries and symmetries ($p=0.038$). A total 50.1% of the pairs were asymmetrical for presence of opacities. Of these, 62.2% scored severity 1 (mild). Symmetry of severity was found for 32.5% of the lesions. Among the pairs of affected teeth, the most frequently observed degrees of lesion severity were mild and moderate, with the exception of lower molars, in which 49% had severe lesions. Conclusions: In this study, MIH lesions were asymmetrical both in presence and severity for all tooth types.

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Keywords: Dental enamel; molar incisor hypomineralization; tooth abnormalities.

Hipomineralización Molar Incisiva: Análisis de la asimetría de las lesiones

RESUMEN

Clinicamente las lesiones de Hipomineralización Molar Incisiva (HMI) no tienen una distribución simétrica variando su severidad inclusive en la misma arcada. Objetivo: Estimar la frecuencia de asimetrías en lesiones de hipomineralización en molares e incisivos permanentes de niños afectados con HMI. Métodos: Tres odontopediatras calibrados (Kappa 0,87) según los criterios de diagnóstico de Mathu-Muju y Wright (2006), registraron presencia y severidad de opacidades en molares e incisivos de pacientes con HMI. En cada paciente se evaluaron 6 pares de piezas dentarias permanentes: primeros molares, incisivos centrales y laterales de ambos maxilares. Para cada una de las piezas se registró el grado de severidad de la lesión (0–intacto, 1–leve, 2–moderado, 3–severo). Para cada par con alguna de sus piezas afectadas, se analizó si presentaba asimetría de presencia (una pieza del par con lesión y la otra intacta), asimetría de severidad (ambas piezas con lesión pero con distinto grado de severidad) o simetría (ambas piezas afectadas con el mismo grado de severidad en la lesión).

Se calcularon porcentajes, I.C. del 95% y χ^2 para las comparaciones. Resultados: La muestra quedó conformada por los 475 pares de piezas dentarias que presentaron lesión entre un total de 1032 pares de piezas analizadas en los 172 pacientes incluidos en el estudio (edad media $11 \pm 2,2$ años, 50% género femenino). El 67,5% (63,1- 71,7) de los pares de piezas dentarias estudiados presentaron relación de asimetría. La relación entre frecuencia de asimetrías y simetrías fue significativa ($p= 0,038$). Del 50,1% de los pares que presentó asimetrías en presencia, un 62,2% mostró grado de severidad 1 (leve) en una de sus piezas. Entre los pares afectados, las lesiones leves y moderadas fueron las más observadas en todos los grupos dentarios, a excepción de los molares inferiores que revelaron un 49% de lesiones severas. Conclusiones: En este estudio las lesiones de HMI presentaron, en su mayoría, algún tipo de asimetría (de presencia o de severidad) en todos los grupos dentarios.

Palabras clave: Esmalte dental; hipomineralización molar incisiva; anomalías dentarias.

INTRODUCTION

Hereditary, environmental and local factors can cause structural defects in the dental enamel of

primary and/or permanent teeth. Depending on the amelogenesis period affected, these defects will be quantitative if such factors act during the protein

matrix secretion phase, or qualitative if they act during maturation or mineralization processes.

Until a decade ago, the three developmental defects most frequently cited in the literature were amelogenesis imperfecta, endemic fluorosis and hypoplasia.

Amelogenesis imperfecta includes a series of clinically and genetically heterogeneous hereditary disorders with low prevalence¹. According to the classification by Witkop, C.J. Jr.², there are 4 types: hypoplastic, hypomaturational, hypocalcified and hypoplasia-hypomaturational associated to taurodontism. In addition to phenotypic criteria, more modern classifications include genetic criteria, molecular defects and biochemical results, when known³. Although Witkop's classification is still the most often used, it is based on phenotype as primary discriminating factor, and inheritance mode as secondary discriminating factor. There is no classification correlating phenotype/genotype, so it has recently been proposed that inheritance should be the primary classification factor⁴.

Dental fluorosis is considered to be a geochemical disease⁵ resulting from excess fluoride intake during the odontogenesis period, which translates clinically, depending on its severity, into white, usually symmetrical opacities with diffuse borders (mild fluorosis), ranging to dark brown stains with enamel erosion (moderate to severe).

Finally, hypoplasias are quantitative enamel defects caused by factors acting on the initial phase of matrix secretion, causing a deficit in the quantity of adamantine structure. They may present as shallow or deep fossae with vertical or horizontal grooves, with partial or total absence of enamel⁶.

Research in recent years, particularly in pediatric dentistry, has focused on Molar-Incisor Hypomineralization (MIH), considered to be an "emerging disease" because it has recently acquired epidemic character⁷. It presents as an anomaly in tissue translucency, with demarcated white/yellow/brown colored areas, without alteration of enamel thickness, which may sometimes disintegrate, giving rapid rise to caries. In contrast to amelogenesis imperfecta and endemic fluorosis, which are considered hypomaturational defects due to high level of residual amelogenins, MIH is typified as hypocalcification with a normal level of residual amelogenins⁸.

Worldwide MIH prevalence calculated from 79 studies in 36 countries is currently 15%⁹. Previous

papers published by our team reported the prevalence of MIH in Buenos Aires City¹⁰ and evaluated preventive strategies with different alternatives¹¹.

The high frequency of MIH and its impact on needs for treatment have made it a silent public health problem, and it is the enamel anomaly that involves highest social cost¹².

Although publications on MIH have increased dramatically, neither its etiology nor the best preventive and restorative strategies are yet clear. Another as yet unresolved issue is that although it is a chronological defect, clinically, MIH lesions are not distributed symmetrically. Within the same tooth type in a single patient, lesions either may not appear or may present different degrees of severity, ranging from mild opacities to post-eruptive breakdown.

The aim of this study was to estimate the frequency of asymmetries in hypomineralization lesions in permanent molars and incisors of children affected with MIH.

MATERIALS AND METHODS

An observational, prospective, cross-sectional study was designed, which included all children with MIH seeking dental care the Department of Comprehensive Pediatric Dentistry at the School of Dentistry of Buenos Aires University and at three private practices in the same area, from March to September 2017, who provided agreement and whose legal guardians provided consent. The study was approved by the Institutional Ethics Committee (FOUBA 26092012-28).

Three pediatric dentists calibrated following the diagnostic criteria of Mathu-Muju and Wright¹³ (2006) (Kappa 0.87) identified presence and severity of opacities on permanent molars and incisors. Six pairs of teeth were evaluated in each patient: first maxillary molars (1.6 and 2.6), first mandibular molars (3.6 and 4.6), central maxillary incisors (1.1 and 2.1), lateral maxillary incisors (1.2 and 2.2), central mandibular incisors (3.1 and 4.1) and lateral mandibular incisors (3.2 and 4.2). For each of the 12 teeth, degree of lesion severity was recorded (0–none, 1–mild, 2–moderate, 3–severe). For each pair, it was determined whether there was symmetry (both teeth affected by the same degree of severity in the lesion) (Fig. 1), asymmetry of presence (one tooth in the pair with lesion and the other without) (Fig. 2) or asymmetry of severity (both teeth with lesions, but with different degrees

of severity) (Fig. 3). Pairs of teeth without clinical lesions were not included. The data were entered into an Excel spreadsheet and analyzed statistically with software R. Percentages, 95% confidence intervals and Chi-Square were calculated.



Fig. 1: Symmetry of presence.



Fig. 2: Asymmetry of presence.

RESULTS

The sample consisted of 475 pairs of teeth with lesions out of a total 1032 pairs evaluated in the 172 patients included in the study (mean age 11 ± 2.2 years, and 50% female).

Of the pairs of teeth studied, 67.5% (63.1 - 71.7) showed asymmetry. Analysis of asymmetrical lesions showed that most asymmetries were due to the presence of unilateral defects, with a total 50.1% of the pairs revealing asymmetry of presence. There was a significant relationship between asymmetries and symmetries ($p=0.038$) (Table 1).

No significant difference was found at 0.05 confidence level between percentage of teeth affected on right side (34.98%) and left side (34.01%) ($p=0.6433$). There was, however a small but significant difference ($p=0.002$) between percentage of affected maxillary (37.69%) and mandibular (31.30%) teeth. Total sample (2064 teeth) was used to calculate and compare these percentages.



Fig. 3: Asymmetry of severity.

Table 1: Number and percentage of symmetries and asymmetries per tooth.

	Cases		Asymmetry Total		Asymmetry Presence		Asymmetry Severity		Symmetry	
	No.	%	No.	%	No.	%	No.	%	No.	%
UM	139	29.3	91	65.5	50	36.0	41	29.5	48	34.5
LM	149	31.4	98	65.8	66	44.3	32	21.5	51	34.2
UCI	81	17.0	51	63.0	46	56.8	5	6.2	30	37.0
ULI	35	7.3	27	77.1	25	71.4	2	5.7	8	22.9
LCI	45	9.5	36	80.0	34	75.6	2	4.4	9	20.0
LLI	26	5.5	18	69.2	17	65.4	1	3.8	8	30.8
Total	475	100	321	67.5	238	50.1	83	17.4	154	32.5
CI 95%				(63.1-71.7)		(45.5-54.7)		(14.1-21.2)		(28.2-36.8)

UM: upper molars; LM: lower molars; UCI: upper central incisors; ULI: upper lateral incisors; LCI: lower central incisors; LLI: lower lateral incisors.

Lower central incisors had the highest percentage of asymmetrically distributed lesions (80%). No significant difference was found in the symmetry/asymmetry ratio between different tooth types ($p=0.95$).

The combinations that included a tooth with mild severity were the most frequent. Mild severity was observed in 62.2% of the pairs with asymmetry of presence and in 55.8% of pairs with symmetrical lesions. Of the pairs with asymmetry of severity, the most frequent combination was mild-moderate (1-2), with 43.4%. Lower molars had 49% severity in pairs with symmetry, being the group of teeth with the highest degree of severity for both symmetrical and asymmetrical lesions (Table 2).

DISCUSSION

Enamel is formed during a defined period of odontogenesis, known as amelogenesis. It is an extremely complex, genetically controlled process, and ameloblasts are particularly sensitive to environmental changes during this phase¹⁴. Dental anomalies are caused by complex interactions among genetic, epigenetic and environmental factors during the period of dental development, which is multifactorial, multidimensional, multilevel and progressive over time, but involves critical periods¹⁵. Even when a specific mutation of a single gene or one important environmental factor has been identified in a patient with a dental anomaly, detailed exploration of phenotype may reveal variations among affected individuals in the same family, between dentitions in the same individual, and even among different teeth

in the same dentition¹⁵. The stage of amelogenesis at which a given tooth germ is at a particular moment in time when the insult occurs is critical for the type and location of the defect. The teeth mainly affected by MIH, though not the only ones, are permanent incisors and first molars. Permanent incisor enamel forms between approximately 3 months and 5 years of age, while first molar enamel forms at about the 8th month of intrauterine life and continues to the age of 4 years. It is therefore believed that the factors causing the disease would act during those periods, with the first 10 months of life being critical¹⁶.

In contrast to hypoplasias which present symmetrically, except when they respond to an identifiable environmental factor (e.g., and infection or trauma to a primary tooth which could cause an alteration in the replacement tooth), in MIH, as shown by the results obtained in the current study, lesions are asymmetrical for both presence and severity. The reason for this is not clear, and the literature contains no paper explaining this situation. With regard to asymmetry of frequency, Padavala and Sukumaran¹⁷ (2018), in a study on only 22 children with MIH, report that teeth on the right side are more affected. This is not consistent with the current study, in which no significant difference was found between sides upon considering a much larger sample. With relation to the presence of disease in upper and lower jaws, our results also contradict the same study, which reports that lower teeth are more affected.

MIH etiology seems to be multifactorial with associated genetic predisposition associated to one or

Table 2: Number and percentage of each possible combination of severity in teeth for each type of asymmetry or symmetry.

	Asymmetry of presence							Asymmetry of severity							Symmetry						
	0-1		0-2		0-3		Total	1-2		2-3		1-3		Total	1-1		2-2		3-3		Total
	N°	%	N°	%	N°	%	N	N°	%	N°	%	N°	%	N	N°	%	N°	%	N°	%	N
UM	22	44.0	18	36.0	10	20.0	50	16	39.0	15	36.6	10	24.4	41	18	37.5	16	33.3	14	29.2	48
LM	24	36.4	21	31.8	21	31.8	66	11	34.4	12	37.5	9	28.1	32	19	37.3	7	13.7	25	49.0	51
UCI	38	82.6	8	17.4	0	0.0	46	4	80.0	0	0.0	1	20.0	5	30	100.0	0	0.0	0	0.0	30
ULI	22	88.0	3	12.0	0	0.0	25	2	100.0	0	0.0	0	0.0	2	7	87.5	0	0.0	1	12.5	8
LCI	28	82.4	6	17.6	0	0.0	34	2	100.0	0	0.0	0	0.0	2	7	77.8	2	22.2	0	0.0	9
LLI	14	82.4	3	17.6	0	0.0	17	1	100.0	0	0.0	0	0.0	1	5	62.5	3	37.5	0	0.0	8
Total	148	62.2	59	24.8	31	13.0	238	36	43.4	27	32.5	20	24.1	83	86	55.8	28	18.2	40	26.0	154

UM: upper molars; LM: lower molars; UCI: upper central incisors; ULI: upper lateral incisors; LCI: lower central incisors; LLI: lower lateral incisors.

more environmental factors acting during a specific period of amelogenesis of a specific tooth. Brook¹⁵ suggests that this may be explained by the multidimensionality of the process of molecular and cellular interactions and their outcomes which occur during the etiology of dental anomalies. The different tooth germs are at different stages of development at a particular time, so an insult during a specific time period could cause different defects in different teeth, depending on the specific formation of that tooth germ.

Symons and Gage¹⁸ claim that in genetic anomalies, all quadrants should be affected in the same way, although severity could increase due to a variation in gene penetration.

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- The current study, as a first approach to the subject, and in order to facilitate interpretation of results, used the Chi-square test for comparisons. A further study could be analyzed using mixed models, to consider possible correlation among teeth in one patient. MIH is a disease that still gives rise to controversies in the literature, especially because its exact etiology is unknown to date. When causal factors are identified, the reasons for asymmetries in lesions will likely be explained.

CONCLUSIONS

In this study, most MIH lesions presented some type of asymmetry (asymmetry of presence or of severity) for all tooth types.

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