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# MOLAR-INCISOR HYPOMINERALIZATION (MIH): REPORT OF A SEVERE CASE

# *HIPOMINERALIZAÇÃO MOLAR-INCISIVO (HMI): RELATO DE UM CASO SEVERO*

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## Abstract

The term *molar-incisor hypomineralization* describes an uncommon pathological entity characterized by the hypomineralization of permanent first molars and commonly affecting permanent incisors. In this paper we describe a case of a 9 years old female patient attended with severe dental sensibility, masticatory and phonetic difficulties. Clinical examination revealed severe enamel structural defects affecting all four permanent first molars and opaque stains affecting permanent incisors, besides caries lesions on posterior teeth and severe osseous basis discrepancy. Radiographic examination evidenced permanent first molars imbrication and shallow lesions on the enamel of these teeth. Therefore, molar-incisor hypomineralization was defined. After fluoride varnish applications, glass ionomer cement restorations and home performed fluoride mouthwash, patient continued to report dental sensibility. Molar-incisor hypomineralization is capable of led the affected patient through morbidity which results on quality of life decrease. Due to severe dentin exposure, therapeutic interventions aiming to reduce dental sensibility are not always effective.

**Descriptors:** Tooth demineralization • Dental enamel • Fluorides • Incisor

## Resumo

O termo *hipomineralização molar-incisivo* descreve uma entidade patológica rara caracterizada pela hipomineralização dos primeiros molares permanentes, frequentemente afetando incisivos permanentes. Neste trabalho é descrito um caso de uma paciente de 9 anos de idade que se apresentou com sensibilidade dental severa, dificuldades mastigatórias e fonéticas. O exame clínico revelou graves defeitos de esmalte estruturais que afetavam todos os quatro primeiros molares permanentes e manchas opacas que afetavam os incisivos permanentes, além de lesões de cárie em dentes posteriores e grave discrepância entre as bases ósseas. O exame radiográfico evidenciou imbricação dos primeiros molares permanentes e lesões superficiais no esmalte desses dentes. Diante desse quadro clínico, foi definido o diagnóstico de hipomineralização molar-incisivo. Após aplicações de verniz fluoretado, restaurações de cimento de ionômero de vidro e bochechos fluoretados realizados em casa, a paciente continuou a apresentar sensibilidade dental. A hipomineralização molar-incisivo proporciono à paciente alta morbidade e redução na qualidade de vida. Diante da grave exposição da dentina, intervenções terapêuticas com o objetivo de reduzir a sensibilidade dental nem sempre são eficazes.

**Descritores:** Desmineralização do dente • Esmalte dentário • Fluoretos • Incisivo

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## Introduction

Amelogenesis is understood as the process which in teeth enamel is developed by mediation of specialized epithelial cells called ameloblasts[1]. This process is didactically classified in two stages; the first stage of amelogenesis is characterized by a protein matrix deposition on a concentric layers pattern, which is followed by the maturation stage, when ameloblasts perform this enamel protein matrix replacement by mineral content, therefore enamel develops into the hardest tissue of human body[1]. During this complex process, external influences might lead to several enamel defects[2]. This wide variability of enamel defects is also classified according to the amelogenesis stage which in ameloblasts are injured[3]. Therefore, enamel hypoplasias represents enamel structural defects such as thickness reduction, by being due injuries to ameloblasts during deposition stage, whereas enamel hypomineralization defects are due to injuries to ameloblasts during maturation stage, which affects the protein matrix replacement by mineral content[2, 3, 4, 5].

Among enamel hypomineralization defects, an interesting pathologic entity is known as molar-incisor hypomineralization (MIH), which was defined by Weerhejein *et al.*[6] (2001) as hypomineralization phenomenon of systemic origin which affects one to four first permanent molars and are usually associated to incisors. Clinically, MIH might be recognized by the enamel presentation of white, cream, yellow to brownish stains, with a crispy and porous "holland-cheese" consistence, always with a well-demarcated boundary between the enamel affected portion and the sound enamel[2,7]. Being a hypomineralized defect, enamel thickness is not altered, however, its resistance to masticatory forces are low when compared to normal enamel, therefore enamel might break down after tooth erupting, creating a false idea of hypoplastic enamel, nevertheless, margins of those disintegrated areas are irregular, whereas in hypoplasia, margins defects are smooth[5,8]. Furthermore, post eruptive breakdown (PEB) leads affected teeth to elevated susceptibility for carious lesions development, high sensitivity to thermal and mechanical stimuli and poor esthetic appearance[8], which, according to Scheffel *et al.*[9] (2014), might be a reason for bullying among children.

As previously mentioned, MIH is considered as a systemic origin disease[10]. Although, studies performed until the moment allow to believe MIH represents a multifactorial condition, which in medical, systemic and environmental factors might be acting additionally or even synergistically to genetic factors[11]. Among systemic factors, nutritional and oxygen supply disorders during pre, peri and postpartum periods are associated to MIH occurrence in children, for example, some authors have indicated prolonged breastfeeding period or oxygen suppression during partum results in MIH[8].

Clinical approach for MIH cases is challenging for dental surgeon once these teeth might be highly sensitive, susceptible to fast dental caries development, limited cooperation of a young child, difficult on achieving anesthesia and recurrent episodes of restorations breakdown[8]. Therefore, available treatment modalities for teeth affected by MIH are extensive, ranging from prevention, palliative, and restorative to extraction of affected teeth in most severe cases[3].

## Case report

Patient NGMS, 09 years old, female gender, presented with main complain of high dental sensibility, masticatory and phonetic difficulty, facial muscular pain, besides dental caries on posterior teeth. On anamnesis patient's mother did not relate any non-nutritive habit by the child, as finger or pacifier. Clinical examination revealed severe discrepancy between patient's osseous basis with appearing mandibular protrusion (Figure 1-A), which in occlusion was present only on first permanent molars (FPMs) (Figure 1-B/C), what lead the patient to permanent "open bite", providing tongue interposition during speaking, pain symptomatology of facial muscles and temporomandibular articulation (ATM), besides alimentary difficulties.

During dental examination, subtle opaque hypoplastic stains were observed on



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permanent incisors, whereas on all four FPMs examiner noticed extensive irregular enamel lesions exposing dentin (Figure 2). FPMs also presented intense pain to mechanical and thermal stimuli. Panoramic radiographic examination was solicited aiming to evaluate more deeply the dental and osseous conditions of the patient, which revealed the imbrication of the FPMs, preventing other teeth to occlude (Figure 3). Radiographic examination also revealed the lesions on FPMs were restricted to enamel, apparently being shallow lesions instead the dentin exposure and intense sensibility (Figure 3).

Based on the pattern of hypomineralization presented and radiographic evaluation, as well as on the related history and symptoms, the diagnosis of MIH was determined. As initial treatment, 4 fluoride varnish applications on the FPMs (1 per week), totalizing a 28 days interval was performed. Patient was also instructed to perform fluoride mouthwash with a NaF 2% solution aiming to reduce sensibility.

For mouth ambient fitness, we also performed shallow restorations with photoactivated glass ionomer cement (GIC) (GC Fuji II LC® - GC America), after local anesthesia of each hemi arc, cleaning of the dental elements and 5 minutes anti-inflammatory (Otosporin®) administration. Finally restorations were covered with GIC glaze, as recommended by the fabricant.

Even after fluoride therapy with varnish, fluoride mouthwash and cavities

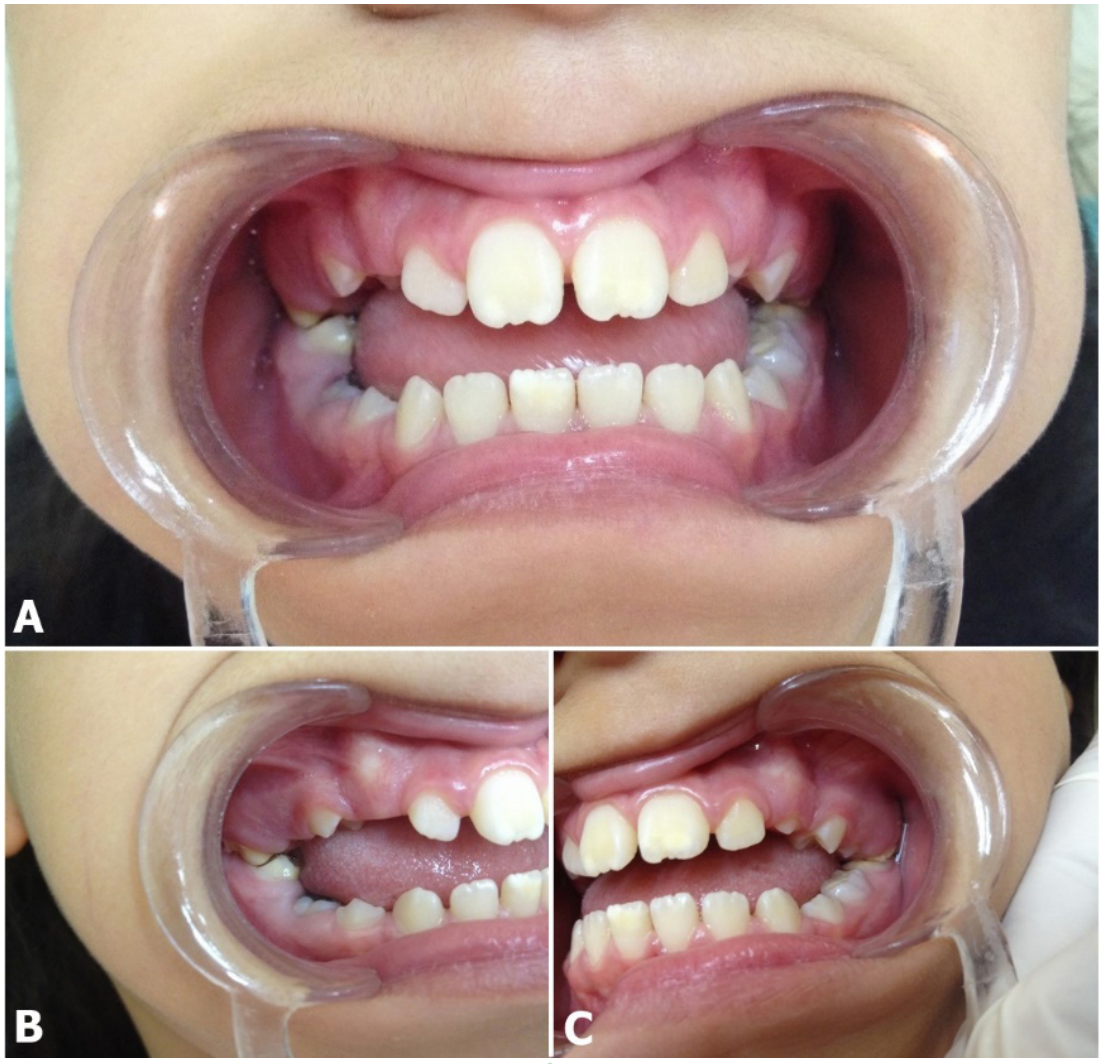


**Figure 1.** *Clinical examination.* **(A)** *Front view showing occlusion only on first permanent molars and apparent mandibular protrusion.* **(B)** *Lateral [1] view showing occlusion only in first permanent molars.* **(C)** *Lateral (left) view showing occlusion only in first permanent molars.*



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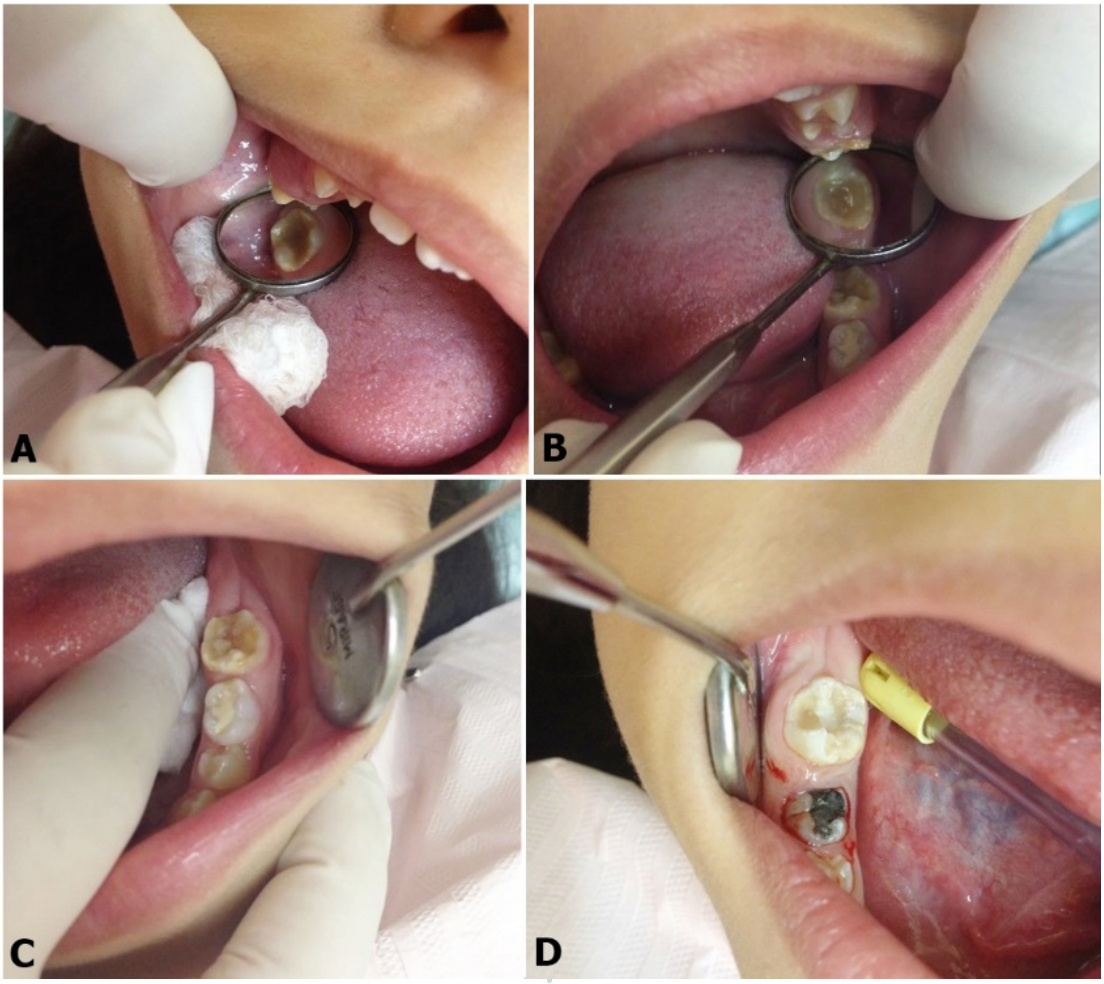


**Figure 2.** *Enamel structure loss with dentin exposure.* **(A)** *Upper right first permanent molars.* **(B)** *Upper left first permanent molars.* **(C)** *Lower left first permanent molar.* **(D)** *Lower right first permanent molar.*



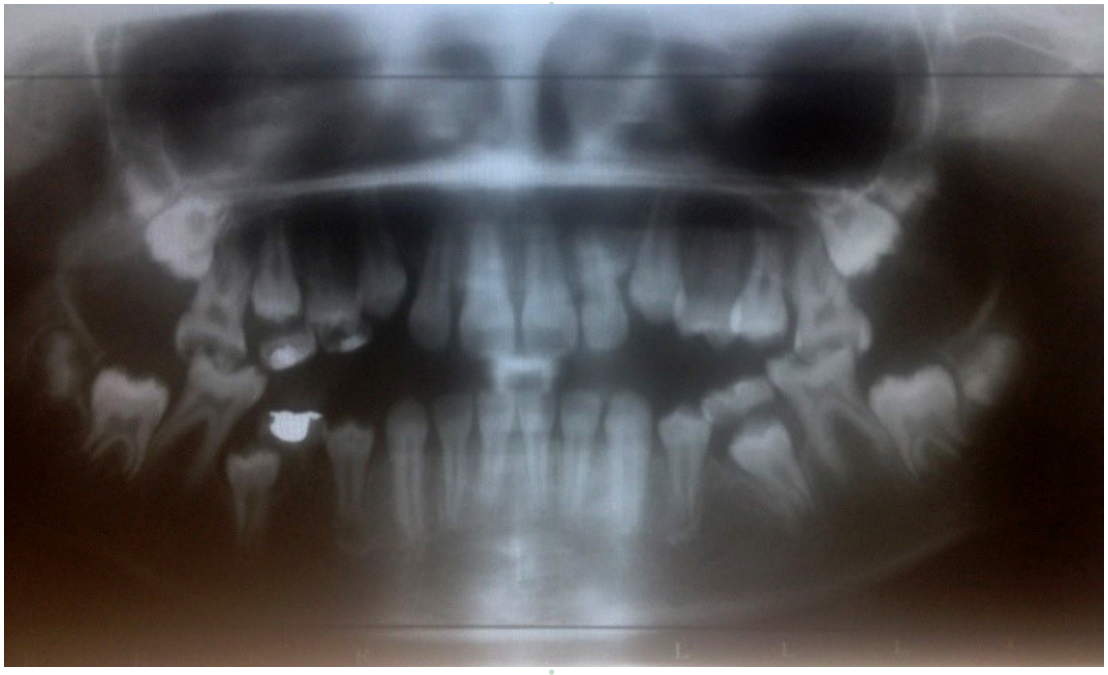

**Figure 3.** *Panoramic radiographic aspect showing apparently shallow lesions on first permanent molars enamel and imbrication of first permanent molars.*

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enclosure, patient kept to relate intense sensibility. Patient is now under clinical follow-up and was related to a maxillofacial surgeon, to determine the correction viability for the osseous basis discrepancy, as well as the preservation of the FPMs affected by MIH on the oral cavity, according to orthodontic and surgical planning.

## Discussion

Clinical importance of adequate knowledge regarding MIH by dental surgeon is not due only to a concrete increased risk for caries lesions development over affected teeth surface, but also to several morbidity experimented by affected patients, as well as high dental sensibility to thermal and mechanical stimuli, masticatory disturbances and esthetical disadvantage[8], which according to Oyedele *et al.*[12] (2015) affects negatively on affected children quality of life. MIH is recognized as hypomineralizations of systemic origin affecting mainly FPMs and incisors[7]. Since we know once formed, enamel structure does not suffer remodeling or composition changes, clearly, defects like these must only be due to injuries during amelogenesis[1]. As mentioned before, enamel hypomineralization defects are led by injuries to ameloblasts during maturation stage of amelogenesis, which is characterized by protein matrix replacement by mineral content[1-3]. MIH clinical manifestation pattern is attributed to the fact that permanent dentition development stage depends on the affected child age, therefore, different teeth are susceptible to developmental disturbances at different times, therefore, hypomineralization defects of FPMs and incisors are due to injuries to amelogenesis during the first years of life, once enamel mineralization begins around birth extending to the fifth year of life for incisors and third year for FPMs[13]. MIH clinical features varies from well-demarcated opacities, white, cream, yellow-brown stains to extensive areas of enamel structural loss present on FPMs and incisors[5,14].

This paper describes a severe MIH case on a 09 years old girl. Clinically, FPMs presented with extensive enamel structure loss with dentin exposure, which was attributed to enamel post eruption breakdown (PEB). This structural defect might be confused with hypoplastic defects; however, hypoplastic defects are characterized by smooth and rounded margins, whereas in PEB defects these margins are irregular[8], what was clearly noticed on FPMs lesions of our patient. Moreover, well-demarcated opacities were noticed over remaining enamel structure of FPMs and incisors, whereas other teeth were not affected, excluding possible diagnosis of *Amelogenesis Imperfecta*[8,15]. Fluorosis hypothesis was excluded once opacities found in fluorosis are mainly diffuse, whereas in MIH like in this case, enamel stains and opacities are usually well-demarcated[8]. Therefore, MIH diagnosis for this case is justified not only by the visual aspect of the lesions, but also by dental affection pattern. Although this case affects a female child, prevalence rates for MIH, which ranges from 8.6% to 10.48%, does not show any consistent statistical gender predilection[16, 17].

MIH etiology remains undefined and controversial; however, multifactorial behavior is well-accepted for this condition development, such as environmental, medical, systemic and genetic factors. Allazzam *et al.*[16] (2014), related most prevalent systemic injuries on first years of life of children with MIH diagnosis were tonsillitis, adenoiditis, asthma, fever and antibiotics intake. Moreover, further authors have described oxygen suppression during birth, nutritional lack during first years of life and medical disturbances suffered by the mother and patient during pre, peri and postnatal periods as possible causes for MIH occurrence[2,5,10]. Although, as MIH is result of injuries which occurred during patient's first years of life[11], to determine which factor might be involved depends on information provided by patient's mother or caregiver, which, in this case, did not provide any information possibly helpful to define an etiologic factor associated to MIH occurrence in this child.

As previously described, MIH is result of amelogenesis maturation stage defects which consists on the protein matrix content replacement by mineral content, therefore, MIH affected teeth enamel is

composed by a protein component more concentrated than in normal teeth enamel, and dentin below hypomineralized enamel also shows a calcium concentration lower than dentin below normal enamel[2,5,8]. Organic matter, as well as protein, is known to have poor acid solubility, therefore, a high concentration of organic matter in hypomineralized enamel might inhibit adequate creation of an etch profile, which compromises adhesion between resin based restorations and affected enamel. Regarding this, restorative therapy with resin materials is not always viable in severe cases of MIH, frequently showing recurrence of restoration loss[2]. For this reason, clinical approach for severe cases like the presented in this paper is usually palliative, aiming dental sensibility reduction or cessation, and preventing caries[3,18]. Instead of fluoride varnish application and GIC shallow restorations to cover exposed dentin have been described as effective for reducing dental sensibility[18,19], in some cases this approach might not show that effectiveness. In this case, patient kept to relate sensibility after varnish application and GIC restoration performed. When success is not achieved with non-invasive therapy, the last choice is to surgically remove teeth with high sensibility[8]; nevertheless due to this patient present a severe discrepancy between osseous basis, this decision will depend on the orthodontic and surgical treatment plan which is about to be established by the maxillofacial surgeon colleague.

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