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Molar Incisor Hypomineralisation in Libyan Monozygotic Twins: A Case Report

***Abstract*—** A systemic abnormality of dental tissue that affects one or more permanent first molars and occasionally the permanent incisors are called Molar Incisor Hypomineralisation (MIH). It is a developmental enamel defect. This case report aims to describe the features, clinical significance, and management of MIH, which was found in a pair of male monozygotic twins. The 8.4 year-old twin Libyan boys, referred as HM and AM, came to our dental clinic with their parents. They were healthy and had no serious concerns. However, both twins reported intense discomfort from the lower permanent molars, sensitive teeth, discoloured teeth, and a history of easily broken teeth. The twins' and their mother's medical and familial histories, as well as the patients' personal information, were meticulously documented to rule out other diseases and identify other contributory variables. According to the interview, the mother had a planned caesarean section to deliver the twins, both were underweight at birth. An intraoral examination revealed that both twins had poor oral hygiene with several dental cavities. Both twins and their parents were given oral hygiene instructions and educated about MIH. All the affected teeth were treated with topical fluoride varnish as the preventive treatment. Early diagnosis and long-term follow-up are crucial to prevent post-eruptive enamel breakdown, caries formation, and tooth sensitivity. Premature molar loss can be prevented with an early detection and treatment of the problem. Since the cause of MIH is still mostly unknown, it is difficult to prevent the illness and create effective treatment plans.

***Keywords*—Developmental enamel defect, discoloured teeth, molar incisor hypomineralisation, monozygotic twins, post-eruptive breakdown, sensitive teeth.**

1 INTRODUCTION

Developmental dental abnormalities are changes in the morphogenesis of teeth brought on by a combination of acquired, developmental, and congenital causes. MIH is classified into five categories, which differ in size, form, number, location, and structure [1-3]. Molar incisor hypomineralisation (MIH) was initially described by Weerheijm et al. in 2001, as a developmental qualitative enamel deficiency, as hypomineralisation of systemic origin of one to four of first permanent molars (FPMs), which is commonly linked to affected incisors [4, 5]. Over the past ten years, MIH has been the subject of greater research. Its aetiology is complicated, involving both hereditary and systemic medical factors. To clarify any additional components and improve the model, more targeted laboratory research and prospective clinical investigations are required [6]. In addition, the history of systemic disorders during the ameloblasts' maturation stages is frequently used to support the diagnosis of MIH, which is mostly based on the clinical appearance of the teeth. In paediatric dentistry, MIH has gained importance in the past 20 years, given the ongoing increase in the perceived incidence and the significant effects on the impacted children and their families [7].

Numerous potential causes have been proposed in the literature, including the potential genetic component of aetiology, which suggests that genetic variation may interact with systemic factors to cause defects in dental enamel. In fact, variations in the genes linked to amelogenesis were also linked to the risk of developing MIH [8]. A few aetiological theories have been put forth to explain MIH. Amelogenesis of the first permanent molars, permanent incisors, and second primary molars begins during the critical period, which lasts from the 28th week of intrauterine life to the initial few weeks of life. A multifactorial pathogenesis with a possible genetic involvement

seems likely, but prenatal exposure to maternal smoking or illness, perinatal factors like low birth weight, premature or prolonged birth, caesarean-section delivery, and complications following delivery, and postnatal exposures such as sixth disease (roseola), medicines, or extended breastfeeding have been suggested [4].

Since its introduction by the European Academic Paediatric Dentistry (EAPD) in 2003, the diagnostic criteria for MIH have undergone modifications [9]. There are many indices available to measure developmental enamel defect (DED), such as developmental defects of enamel (DDE) index, modified DDE, enamel defect index (EDI), and modified Clarkson and O'Mullane DDE index, the most used indices are modified DDE and EAPD criteria. On the other hand, a new MIH severity scoring system (MIH-SSS) categorisation was created in 2020 [10]. The reliability of this new classification has only been the subject of one study, though. The modified DDE index, however, is not always able to accurately diagnose hypomineralisation brought on by fluorosis, MIH, or enamel abnormalities. As a result, modified DDE is not regarded as a sufficient MIH index [11]. Depending on its severity, MIH can manifest clinically in different ways. The mildest involvement is represented by white or creamy opacities, while a higher degree of hypomineralisation is indicated by yellow-brown opacities. After eruption, lesions may manifest as uneven patches of post-eruptive disintegration, which can be confused with hypoplasia, particularly when they come into contact with the opposing teeth. On surfaces that are not typically susceptible to caries, the initiation of caries can result in the development of atypical caries, frequently when neighbouring teeth are free of cavities [12].

MIH's clinical characteristics include soft, porous enamel, flaws in the enamel (i.e. appear as white, yellow, or brown), a discernible difference between clinically injured and unimpacted enamel, tooth sensitivity and enamel loss (post-eruptive enamel disintegration). The term 'hypomineralised second primary molars (HSPM)' has been used to describe similar lesions in the second primary molars [13]. HSPM, atypical caries pattern, and incisor abnormalities often do not cause enamel loss unless the incisal edge is impacted. Enamel hypoplasia is more common in MIH patients, and these abnormalities appear to be caused by the same threatening factor acting at various amelogenesis phases [14]. Hypomineralisation of the molars and incisors can be confused with several different disorders. Accurate diagnosis and the best treatment of patients with MIH depend on knowing the major characteristics that set it apart from diffuse opacities, hypoplasia, amelogenesis imperfecta, and carious white spot lesions [15]. It is crucial to consider the following differential diagnoses of developing enamel abnormalities while making the diagnosis of MIH.

1.1 Amelogenesis Imperfecta

A diverse collection of hereditary disorders marked by widespread enamel developmental abnormalities that impact both the quantity and quality of the enamel. In addition to being hypoplastic, enamel may exhibit hypomaturation, hypoplastic with taurodontism, and hypocalcification. Hypoplasia in enamel pits and grooves is an example of a quantitative flaw that results in decreased enamel thickness. Additionally, patches of lacking enamel may be present [16].

1.2 Fluorosis

Fluoride consumption during enamel growth is the cause of diffuse opacities, known as fluorosis. White spot lesions are the clinical indicator of dental caries that appear early. These lesions typically appear in areas of the tooth's enamel where plaque has accumulated, such as the gingival edge.

1.3 Hypomineralisation due to trauma

Concussion, dislocation, luxation, intrusion, or extrusion of the primary tooth are examples of possible sequelae of periodontal trauma [17].

Diagnosis of MIH and enamel hypoplasia can be made by evaluating the panoramic radiographs and intraoral photographs. The identification of hypoplasia was carried out by observing the presence of a quantitative defect in the tooth enamel with regular and smooth edges. Patients initially diagnosed with MIH via intraoral photographs would be clinically reassessed to confirm the diagnosis, following the criteria defined by the EAPD and modified by Ghanim [14, 15]. MIH is a tooth translucency abnormality, characterised by a demarcated opaque area on the affected tooth surface, distinct from

surrounding sound enamel, ranging from creamy white to yellowish brown [18]. Clinically, the hypomineralised regions that impact the incisors and FPMs present as well-defined opacities. These distinct regions, which can range in hue from white to yellow to brown, are anomalies in the translucency of enamel. Due to the masticatory forces that the teeth undergo, they are frequently linked to post-eruptive enamel deterioration in the molars and are more severely affected than the incisors [5, 19, 20]. Nonetheless, a characteristic of MIH is the modification of enamel, which can range from diffuse opacity to hypoplasia and/or hypomineralisation of enamel and dentine. The teeth that suffer the most are usually the FPMs and permanent canines [21]. Early detection will help prevent such dental complications and maintain the tooth structure for social and aesthetic reasons. Amelogenesis imperfecta, dentine dysplasia, and dentinogenesis imperfecta are examples of anomalies that can be symptoms of a syndrome and cause severe decay and tooth sensitivity [22].

This case report's objective is to discuss the characteristics and treatment of MIH in two healthy males of monozygotic twins.

2 CASE REPORT

Twin Libyan boys, referred as HM and AM, aged 8.4 years, came to the dental clinic with their parents. They were in good health and had no serious concerns. However, both twins complained of sharp pain from the lower permanent molars, discoloured teeth, a history of easily broken teeth, and sensitive teeth. The patients' personal data, as well as the twins' and their mother's medical and familial histories, were meticulously documented to rule out other possible diagnoses and identify potential contributing variables. From the interview, it was known that the mother gave labour through a caesarean section (planned C-section) and the twins had low birth weights. During an intraoral examination, it was discovered that both twins had several dental cavities, poor oral hygiene, plaque accumulation on the labial surface of their anterior teeth, and bleeding when probed. The first twin (HM) had a history of respiratory disease and frequent antibiotic treatment during the first three years of life. His oral examination showed that the upper left FPM (tooth #26) and both lower FPMs (teeth #36 and #46) suffered from post-eruptive enamel breakdown (PEB) while the upper right FPM (tooth #16), upper left second premolar (tooth #25), upper right central incisor (tooth # 11) and lower left central incisor (tooth #31) had whitish demarcated opacities. HM also had HSPM of tooth #55. All these clinical characteristics are shown in Figures 1 and 2.

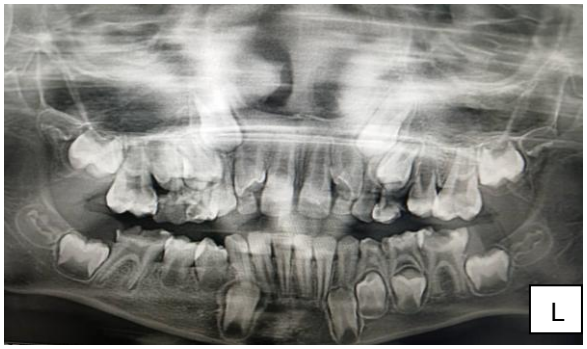

Figure 1: Digital panoramic radiograph of the first twin (HM) showing PEB at teeth #26, #36 and #46 and white demarcated opacity at tooth #25.

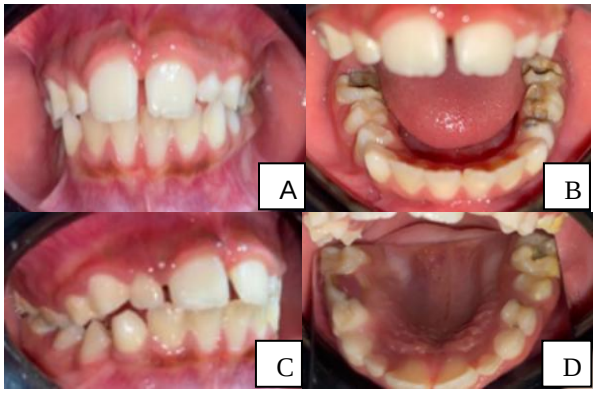

Figure 2: The first twin (HM). (A) The frontal view shows white demarcated opacities at teeth #11 and #31. (B) The mandibular view shows PEB of teeth #36, #46, and HSPM of tooth #75. (C) The lateral view shows the loss of posterior vertical dimension due to PEB of teeth #46, #36 and #26. (D) The upper view shows PEB of teeth #55, #26, and early eruption and white demarcated opacity at buccal cusp of tooth #25.

The second twin (AM) had a history of hernia surgery at the age of two years, with fair oral hygiene and plaque accumulation on anterior teeth. In addition, AM had early eruption of upper right first premolar (tooth #14), PEB of teeth #36 and #46, and HSPM of teeth #55, #65, #75 and #85, and reduced posterior vertical dimensions as shown in Figures 3 and 4. Both twins were diagnosed with MIH based on the clinical assessments, the associated history and

symptoms, and the pattern of hypomineralisation that was displayed in the oral cavity. Firstly, both twins and their parents were given oral hygiene instructions and educated about MIH. Then all the affected teeth were treated with topical fluoride varnish (Duraphat, Colgate Oral Care, Sydney, Australia) as the preventive treatment. In addition, the twins were instructed to continue brushing two times daily with fluoridated toothpaste. The twins will be reviewed regularly to monitor the signs and symptoms.

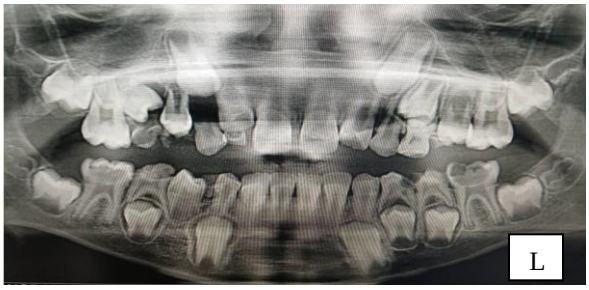

Figure 3: Digital panoramic radiograph of the second twin (AM) showing PEB of teeth #36 and #46.

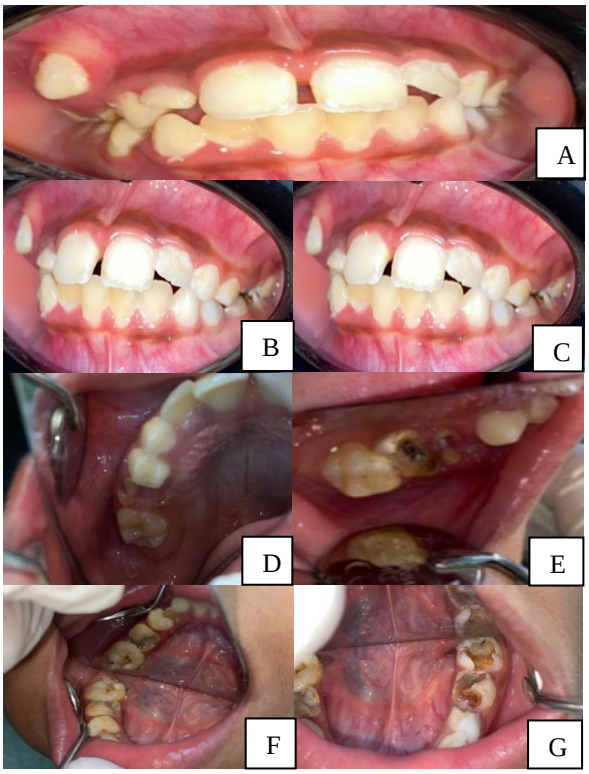

Figure 4: The second twin (AM). (A) The frontal view shows plaque accumulation on anterior teeth. (B) The lateral right view shows an early eruption of tooth #14 and reduced posterior vertical dimension. (C) The lateral left view shows reduced vertical dimension. (D) The upper right view shows PEB of tooth #55. (E) The upper left view shows PEB of tooth #65. (F) The lower right view shows PEB of teeth #46 and #85. (G) The lower left view shows PEB of teeth #36 and #75.

3 DISCUSSIONS

This case report presented monozygotic twins that were born at term, via caesarean section delivery with a low birth weight. The first twin (HM) had a history of respiratory disease and frequent antibiotic treatment during the first 3 years of life. The second twin (AM) had undergone surgery for a hernia when he was two years old. The diagnosis of MIH was confirmed from the clinical characteristics of the affected molars and incisors in combination with the medical history, which could have been a contributing factor. The diagnosis of MIH should be confirmed by clinical examination of clean and wet teeth, ideally after the age of eight, when all permanent incisors and first molars will have mostly erupted, and at least one FPM must be affected, according to Weerheijm's 2003 recommendations [20]. Patients with MIH are more likely to experience health issues in their first four years of life. A similar finding was reported in two monozygotic male twins of 11 years old, the first twin showed significant MIH and white opacity in his teeth. The second twin exhibited a moderate MIH. Their mother took antibiotics during pregnancy and delivered them via caesarean section. The twins were treated with antibiotics after episodes of amygdalitis and high fever. However, respiratory issues only affected the first twin [23].

Children with MIH have a significant prevalence of HSPM and hypomineralised incisors. Compared to the children who received mild involvement, children who received intense involvement were more likely to experience hypersensitivity, dentine caries, and damaged incisors [24]. MIH should be diagnosed promptly and accurately; secondary and tertiary prevention strategies should also consider the multifactorial nature of the aetiology of caries; and treatment and management options for moderate and severe forms of the disease should consider the prognosis based on established risk factors. MIH and HSPM require early diagnosis. Delineated opacities on erupting permanent incisors and HSPM can be used as MIH predictors [24], though not solely. HSPM and/or MIH screens should be performed on children who show more than one of the above aetiological factors. The dental team must provide more frequent dental exams and preventive advice to children with MIH

or HSPM, particularly when the first permanent and second primary molars erupt [25]. However, research on age-specific assessment indicates that the prevalence of MIH increased from 6% to 14% between 2000 and 2010 [7]. It is preferable to detect hypomineralised enamel of the relevant teeth and diagnose MIH when all first molars and incisors have fully erupted, which should happen around the age of eight [26].

MIH most likely results from the combination or cumulative action of genetic and/or environmental factors. Besides, MIH's development may be influenced by heredity [27]. In addition, MIH's incidence may be influenced by the rs13058467 variant of the tubulin tyrosine ligase-like 12 (TTLL12) gene [28]. Additionally, the rs3796704 variant of the enamelin (ENAM) gene was found to have an impact on MIH's incidence [29]. Genetic research has expanded the possibility of identifying the probable impacts of amelogenesis-related gene variations on the development of MIH [30]. Recently, it has been shown that amelogenesis may be impacted by immune response gene variations. It is possible that individual gene variations contribute to the development of MIH, which most likely happens because of environmental conditions [31]. Nevertheless, MIH may be identified while the FPMs are erupting. An early identification could reduce the extent and severity of PEB as well as the elevated risk of dental caries and hypersensitivity in the future [32]. Children may experience psychosocial effects from MIH. According to a study in the UK involving 3233 kids, the frequency of MIH was 15.9% [17]. In Brazil, 167 pairs of twins, aged 8 to 15, involving 94 monozygotic and 73 dizygotic twins, were examined. Although there was more agreement in the incidence of MIH between monozygotic twin pairs than between dizygotic twin pairs, this indicates a genetic component to the illness, whereas environmental factors like family income and the presence of haemorrhage during delivery were also linked to MIH occurrence [33].

A study of 1405 Saudi children found that mandibular teeth were more affected by MIH than maxillary teeth, and maxillary teeth were more involved in the permanent central incisors group. MIH affected all four molars, with mild to moderate severity [34]. Children who had been exposed to antiepileptic medications during pregnancy only developed enamel hypoplasia and diffuse opacities in their primary dentition; this observation can be explained by the fact that primary teeth develop during pregnancy. There were no variations in the prevalence of yellow opacities between the two groups; however, white opacities were prevalent in the exposed children's primary and permanent dentition [35].

Children who have teeth damaged by MIH typically also have incisors that are affected, which can cause dental pain, hypersensitivity, and aesthetic issues. To reduce enamel deterioration and pulpal involvement, early detection and treatment are crucial for molars affected by MIH. The goal of remineralisation therapy is to create a mineralised surface layer, and it should begin as soon as the damaged surface is accessible. Topical fluoride can remineralise enamel, lessen sensitivity, and increase resistance to demineralisation when it is administered as varnishes or gels. In dentistry, fluoride was the first remineraliser agent [36]. Preventive measures such as fissure sealants are recommended on FPMs with mild MIH with no evident enamel breakdown or sensitivity. The affected teeth require regular follow-up to monitor the retention of the sealants [5, 37]. FPMs are capable of post-eruptive disintegration very quickly. Children may exhibit brown or "cheesy colour teeth," sensitivity, discolouration concerns, and in certain situations, accelerated caries advancement. Close coordination between the general dental practitioner, paediatric dentists, and orthodontists is necessary for the comprehensive management of FPMs [17].

For individuals with enamel abnormalities, specifically pre-eruptive lesions, microabrasion is a minimally invasive procedure that is both safe and effective. Compared to restorative procedures, microabrasion is less damaging and permits good aesthetic results with negligible postoperative sensitivity. Even though this method has several drawbacks, it can be improved by combining it with other therapeutic modalities [38]. Managing children with these defects is tough because of the significant pain management issues with these teeth, which are brought on by the exposed dentine's heightened sensitivity and the pulpal cells' subclinical inflammation from the porosity. These deficiencies cause these kids to have more behavioural issues [39, 40]. Depending on the extent of the lesions, the patient's age, the ability to control moisture, and oral hypersensitivity,

restorative techniques might range from glass ionomers or resins to full-coverage crowns. After a thorough assessment of the possible effects extraction may have on the occlusion and long-term growth and development (such as the presence of the second or third permanent molars), extraction may be the best course of action in extreme situations. Sealants or resin restorations are frequently the best course of action for discoloured molars with little to no enamel erosion [41, 42]. By eliminating proteins and improving the resin's ability to penetrate the etched enamel, deproteinisation with sodium hypochlorite (NaOCl) can help restore the enamel's natural colour and may also assist in strengthening resin bonds. Deproteinisation can be achieved by applying 5% NaOCl to the enamel surface for 60 seconds, followed by rinsing and etching, like the traditional sealants or resin placement. Glass ionomers are frequently the most effective initial treatment for molars with severe MIH that are linked to significant enamel loss and extreme sensitivity [43].

The EAPD firmly supports the use of all available treatment options for the MIH-affected teeth, considering the need for a painless and successful treatment plan as well as the paediatric patient's overall health on all levels; oral, dental, medical, and social [6]. Early identification, pain management, restoration, preventative measures, and follow-up observation are all essential to the care of these situations. Conversely, being detected late until the maturity age will result in larger tooth wear, necessitating more extensive dental treatment, involving more time, and more money. The extension and severity of the lesion are important factors in the treatment planning, either by using adhesive restoration with the use of the enamel layer or by crown coverage [16].

4 CONCLUSION

MIH has received particular attention in therapeutic practice, and aetiological information about this illness is crucial. Clinical results involving twins indicate that ameloblasts are specifically impacted during their developmental phase, which encompasses a variety of factors. Prenatal and perinatal complications do not play a decisive role in the development of MIH; however, they have a potential genetic susceptibility to the condition. The cause-and-effect linkages must be verified by prospective observational studies employing a population sample that includes information on the final three months of pregnancy till the eruption of permanent teeth. An early, precise diagnosis and long-term follow-up are crucial to prevent complications such as post-eruptive enamel degradation, caries formation, and tooth sensitivity. The extent and severity of the damaged enamel determine the different management options. Therefore, patients with MIH need to maintain good oral hygiene.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

DECLARATION OF PATIENT CONSENT

For the clinical data to be published in the journal, the parents of the twins had signed the consent forms.

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