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Management of Enamel Defects: A Case Report of Identical Twins with Enamel Defects of the Permanent Dentition

Abstract: Enamel defects have a wide range of aetiology and can be challenging to diagnose. Largely, the management of enamel defects is determined by the severity. As most patients with enamel defects present at a young age, conservative treatment is a priority. Nevertheless, indirect restorations have a role in restoring severely broken down teeth. This article discusses the diagnosis and management of identical twins with molar-incisor hypomineralization (MIH). Many of the techniques discussed can be applied to a wide presentation of enamel defects and are not limited to the management of MIH.

CPD/Clinical Relevance: The restorative management of enamel defects can be challenging but priority should be given to conservative treatment techniques for young patients.

Dent Update 2017; 44: 1049–1056

Enamel development may be affected by intrinsic or extrinsic factors. These factors can result in defects of enamel structure (hypoplasia) or composition (hypomineralization) (Table 1). Clinical presentation often involves a combination of hypoplasia and hypomineralization which may be present in varying degrees of severity across the dentition.

Diagnosis of these defects is often challenging and requires a thorough history, clinical and radiographic assessment. The differential diagnoses of enamel defects are described in Table 2.

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Diagnosis

Cases 1 and 2 show the dentition of identical twins with enamel defects (Figures 1 and 3). No aetiological factors were identified in the history taken from the patients or their mother.

Type of enamel defect

The white mottling, brown staining and heavily broken down mandibular molars represent an array of enamel defects including molar-incisor hypomineralization (MIH), chronological hypoplasia and amelogenesis imperfecta (Table 2). Specifically, there are hypomineralized patches, pitting and severely broken down first molars.

Distribution of defects

In both cases there appears to be a chronological pattern to the distribution of defects (Figures 1 and 3). The first molars, incisors and canine tips are affected as well as there being associated white snow-capped premolars and second molars. The lower incisors, however, appear to be randomly affected.

Differential diagnosis

As no history of systemic disturbance or childhood illness could be identified from the history, a differential diagnosis of MIH or amelogenesis imperfecta (AI) was reached, based on clinical findings.

Two key points led to the diagnosis of MIH over that of AI. The first being the severity of the breakdown of the first permanent molars relative to the mild defects affecting the incisor teeth, and the second

Enamel Defects	Description
Hypoplasia	Deficient matrix volume – quantitative
Hypomineralization	Poor mineralization – qualitative
-Hypomaturation	Mild changes
-Hypocalcification	Severe defects

Table 1. Defects of enamel structure (hypoplasia) or composition (hypomineralization).

Differential Diagnosis of Enamel Defects	Description
Amelogenesis imperfecta (AI)	-Genetic (possible positive family history) -Affects all/most of primary and permanent dentition -Varied penetrance and presentation across the dentition -May be associated with systemic conditions
Molar-incisor hypomineralization (MIH)	-Various suspected aetiology -Affects 1–4 first permanent molars and permanent incisors -Appears to have chronological pattern -First molars may show rapid breakdown and caries development -Lesions vary (incisors affected less severely)
Dental fluorosis	-Caused by excessive fluoride intake during tooth development -Severity dependent on dose of fluoride -Lesions vary (white opacities, dark discoloration, hypoplastic defects) -Often shows a chronological pattern representing highest fluoride intake
Chronological enamel hypoplasia	-Systemic upset during *in utero,* neo-natal or childhood periods -Chronological pattern representing systemic illness

Table 2. The differential diagnoses of enamel defects

being the random pattern of defects seen on the lower central incisors.[1] These are typical features of MIH.[2]

These cases do additionally demonstrate mild hypomineralization of the premolars and second molar teeth (snow-capped cusps) and possible hypoplasia of the lower incisors where there are pitting defects. These features would be more consistent with a form of AI, however, AI typically affects the whole dentition fairly evenly without apparent chronological patterns, which contrasts with this clinical presentation.[3]

Interestingly, Vieira and Kup have reported hypomineralization of the premolars and second molars in MIH, extending the traditional description.[2] Hypomineralized enamel may undergo post-eruptive breakdown, and present as a quantitative defect, confusing the clinical picture (Figure 1).

These cases demonstrate the difficulties in making a diagnosis by clinical features alone, given the complex multifactorial disease processes involved. Histology of extracted teeth or genetic testing may help confirm aetiology. The implications of this should be dealt with sensitively and a referral for genetic counselling may be helpful. Otherwise, aetiology is likely to have little impact on clinical management and, although this article focuses on MIH, the treatment modalities are transferable to other enamel defects, depending on the severity.

Management

All-ceramic lithium disilicate indirect restorations were provided in Case 1 (LR6) and Case 2 (LL6 and LR6) (IPS e.max® Press, Ivoclar Vivadent, Leicester, UK). To conserve tooth tissue, an onlay prep design was carried out on the LR6 in Case 1 (Figure 2). In case 2, failing crown margins of the LR6 and LL6 were diagnosed. The LR6 and LL6 full coverage crowns were removed and the teeth were restorable allowing replacement crowns to be provided (Figure 4). In order to achieve the most aesthetic result for the patient, clinical photographs with and without the selected shade tab were provided to the dental technician. The detail of the adjacent teeth appearance including 'snow-capping' was incorporated into the final restorations.

Before cementation, the fitting surfaces of the restorations were etched with 5% hydrofluoric acid etching. The teeth were etched for 15 seconds with 37% phosphoric acid and rinsed. Bonding agent (Scotchbond™ Universal) was applied to the internal aspect of the restoration and to the dried tooth surface with agitation for 20 seconds, followed by air drying for 5 seconds. A thin layer of resin-based luting cement (Rely X™ Ultimate, 3M ESPE, Loughborough, UK) of matching shade was applied and the restoration seated. After the excess cement was removed, 20 seconds light curing completed cementation.

In Cases 1 and 2, direct composite restorations were provided on the affected incisors. The teeth were isolated; abraded with pumice and water; etched with 37% phosphoric acid for 20 seconds; bonding agent (Optibond™ Solo Plus, Kerr, Peterborough, UK) was applied and agitated for 10 seconds; the bonding agent was air thinned for 3 seconds followed by light curing for 20 seconds. Freehand incremental composite placement (Herculite XRV™ Ultra, Kerr, Peterborough, UK) was carried out to cover the enamel defects (Figures 2 and 4). In these cases, the patients did not wish to close the diastema spacing and wished to keep tooth shape and size the same. It is important to establish the patient's desires early on as this may differ from the clinician's or parent's 'ideal aesthetics'.

Discussion

Molar-incisor hypomineralization

Molar-incisor hypomineralization (MIH) has been defined as 'hypomineralization of systemic origin of one to four permanent molars frequently associated with affected incisors'.[4,5] Demarcated enamel opacities in the first permanent molars are common in many child populations, with a prevalence ranging from 3.6%–25%.[6] The first permanent

Figure 1. Case 1: **(a)** Affected incisors reveal brown/white discoloration and the enamel surface is pitted. **(b, c)** The canine tips are involved. **(d)** The mandibular first molars have large failing restorations with recurrent caries. **(e)** The patient was mainly concerned with the discoloration of the anterior teeth.

Figure 2. Case 1: **(a–c)** UR1,2,3, UL1,2,3 and LL1,2,3 discoloration was masked with the addition of direct composite restorations. Further staining discoloration was removed with ultrasonic scaling and polishing. **(d)** The LL6 was deemed unrestorable and extracted. A partial coverage e.max onlay was provided on the LR6. **(e)** The patient was happy with the aesthetic outcome without closing the diastema spacing.

molars are particularly affected, while the permanent incisors and sometimes the cuspal parts of the canines can be associated, usually to a lesser extent (Figures 1 and 3).[7] The degree of disturbance can vary between creamy white spots with a hard, well mineralized surface to a yellow-brown discoloration and enamel breakdown (Figures 1 and 3).[7]

Aetiology of MIH

Williams *et al* describe hypomineralization as being related to disturbed resorptive potential of ameloblasts and proteolytic enzyme inhibition.[8] This leads to protein retention (particularly amelogenin) and interference with crystal growth and enamel maturation.[8] If the primary teeth are affected there is a higher chance that the

Extrinsic Factors
Antibiotics Problems during pregnancy (ie oxygen starvation) Malnutrition Environmental conditions Exposure to polychlorinated biphenyl (PCB)/Dioxins
Intrinsic Factors
Low birth weight Perinatal complications Coeliac disease Respiratory tract infection Childhood illnesses Genetic

Table 3. Potential aetiology for molar-incisor hypomineralization.

permanent dentition will be affected.[9]

Various intrinsic and extrinsic causes for MIH have been suggested within the literature (Table 3). The use of antibiotics are commonly referred to in the literature with particular note of amoxicillin in the first year of life.[10] Despite frequent reports of association, there is no convincing evidence to support that antibiotics cause MIH.[11] As antibiotics are often prescribed to treat disease, it is often difficult to determine whether the antibiotics or disease are responsible.

Clinical studies have shown association between the levels of polychlorinated bisphenol (PCB) exposure and prevalence of enamel defects in the permanent dentition.[12] As good electrical insulators, PCBs were used pre-1986 in electrical capacitors and transformers and enter the body through direct contact, inhalation of their fumes or ingestion of food contaminated by PCBs. An animal study in rats also demonstrated the association of enamel defects after exposure to a similar compound (Tetrachlorodibenzo-p-dioxin), suggesting that these compounds interfere with enamel mineralization.[13]

Studies have shown that premature birth with a low birthweight has been associated with an increased level of hypomineralization and hypoplasia in the permanent dentition.[14,15] One study of 32 premature children demonstrated a prevalence of 83% enamel defects in the permanent dentition compared to 36% prevalence in 64 children with normal birth weights.[15] The severity of the enamel defects increased with a lower birthweight.[14]

It is now well recognized that coeliac disease is associated with oral manifestations including enamel defects, recurrent aphthous ulceration and delayed eruption of teeth.[16] One study found that 52 children with coeliac disease were found to have significantly higher dental enamel defects of 61% (compared to 21% in 52 control children).[17]

Children with poor general health and systemic conditions are more likely to have developmental defects of enamel.[18] Conditions common in the first 3 years, such as upper respiratory diseases, asthma, otitis media, tonsillitis, chicken pox, measles and rubella, appear to be associated with MIH.[19] In a retrospective study of 21 children with MIH, 67% had suffered from bronchitis, asthmatic bronchitis, pneumonia, and upper respiratory tract infections.[20]

A genetic link to the cause of MIH has been suggested[2] and investigated; however, published studies are significantly underpowered and therefore it is not possible to draw definitive conclusions.[21] A 2009 and 2010 review claimed that there was insufficient evidence to determine the aetiology of MIH.[22,23] Many studies are limited by their retrospective nature, which relies on the memory of parents or children.

Preventive Measure	Advice
Toothbrushing	-Two times a day for 2 minutes -Spit out excess but don't rinse after brushing -Once last thing at night
Fluoride toothpaste	-Concentration: 1500 ppm (if under 6 years 1000–1500 ppm) -Pea-sized amount (Smear-sized amount if under 6 years)
Fluoride toothpaste (active caries)	-Over 10 years: 2800 ppm prescription toothpaste -Over 16 years: 2800–5000 ppm prescription toothpaste
Fluoride mouthwash (7+ years)	-Concentration: 0.05%NaF^-/220 ppm -Use at a different time from brushing
Topical fluoride application	-2.2% NaF^-/22 600 ppm -2 or more professional applications a year
Diet advice	-Reduce frequency and amount of sugar-containing drinks and foods -Diet analysis to assist identifying sugar-containing foods -Recommend sugar-free medications -Advise use of healthy, sugar-free alternative snacks
Fissure sealants	-Seal permanent molars with resin or glass ionomer sealant -Revise and replace on signs of deterioration
*Please note that some of the above recommendations apply to high risk patients based on their caries risk profile. The risk of dental fluorosis and caries must be weighed up. The full details of recommended guidance are found in the *Delivering Better Oral Health: An Evidence-based Toolkit for Prevention* 3rd edn, 2014.[25]	

Table 4. Preventive and interventional treatment.

Management strategies for MIH

Molars

Enamel defects affecting the molars are often associated with pain and sensitivity, making oral hygiene measures challenging, even when the enamel is intact.[6] As the enamel structure is more fragile, caries can develop more easily and rapidly. Challenges in anaesthetizing severely affected teeth are common.[6] Of children with MIH, 97% were found to have their first molars

Figure 3. Case 2: **(a–c)** The extent of discoloration is less severe, while matching the pattern of presentation as seen in Case 1. **(d)** The mandibular first molars had failing metal ceramic coverage restorations with sound radiographic and clinical root canal treatment. **(e)** Again the patient was mainly concerned with the discoloration of the anterior teeth.

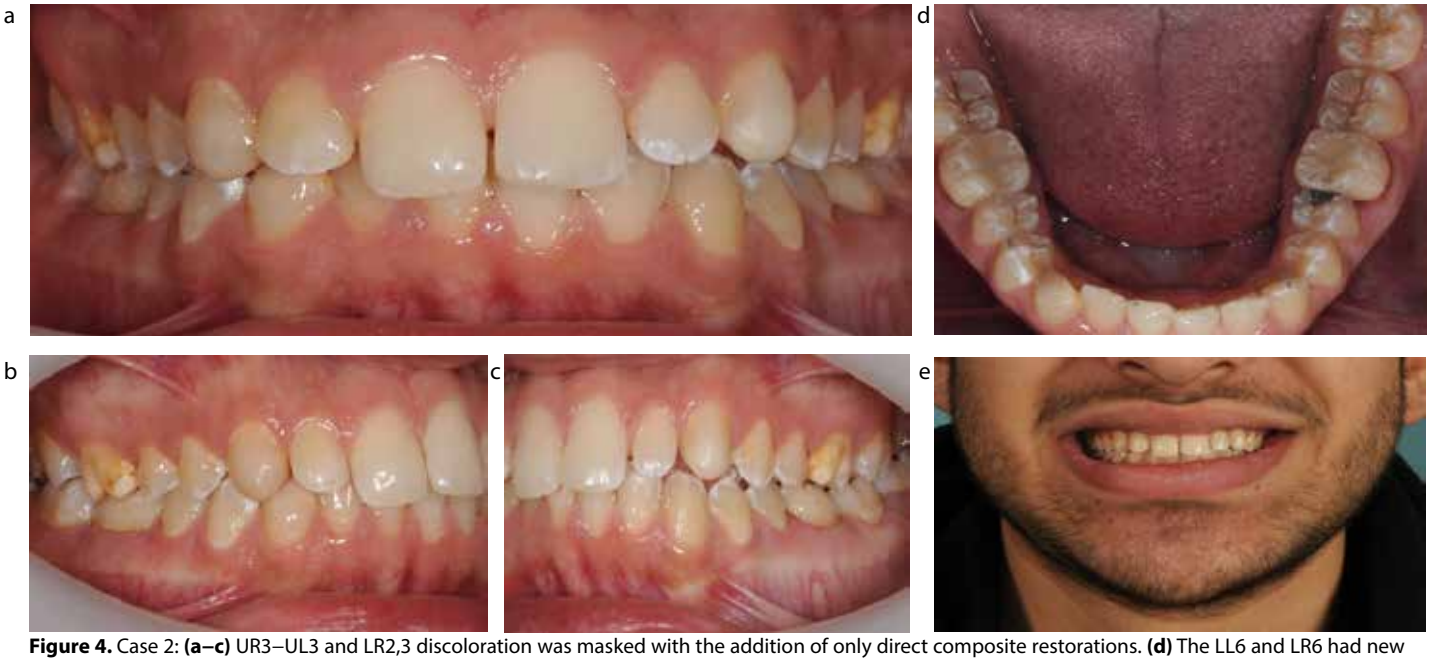

Figure 4. Case 2: **(a–c)** UR3–UL3 and LR2,3 discoloration was masked with the addition of only direct composite restorations. **(d)** The LL6 and LR6 had new core restorations provided with direct composite and indirect full coverage e.max crowns were provided with characteristic mottling added to the cusp tips. Photographic records were sent to the technicians. **(e)** The patient was happy with the aesthetic outcome without closing the diastema spacing.

restored and 28% had one or more extracted.[24] Of children without MIH, 22% experienced restorations in first molars.[24] The majority of the restored teeth in children with MIH were repeated due to loss of restoration, caries or further breakdown.[24] On presentation, patients may have high levels of anxiety and dental fear due to multiple previous treatments and possible inadequate anaesthesia.[24] Cold air and water may cause sensitivity in opposing/contralateral dentition which are not anaesthetized. In addition, children may avoid brushing these teeth due to associated sensitivity, thereby exacerbating the caries progression.

If an erupting molar shows signs of hypoplasia, the child should be closely monitored until the eruption of all

the first permanent molars. Preventive and interventional treatment should be initiated, where appropriate. Oral hygiene instruction, diet advice, toothpaste advice, topical fluoride application and fissure sealants should be considered (Table 4). Where first permanent molars show signs of MIH, parents should be warned about the risk of affected molars and incisor teeth including discoloration. The elevated risk of caries in the molar teeth should be discussed. Parents should be reassured that while the incisors may be affected, presentation is unlikely to be as severe and that tooth loss of incisors is very unlikely.

Adhesive restorations are ideal for affected first molars to minimize sound tooth tissue loss in an immature tooth. The restorations should aim to finish on sound non-affected enamel, however, in cases of severe enamel defects, this is often hard to determine. There may be deterioration of the marginal enamel due to post-eruptive breakdown. Regular repair or replacement of these restorations is often required over time to avoid caries development.

Where teeth are significantly broken down, medium term options include placement of prefabricated metal crowns, which provide a good marginal seal.[26] This may allow definitive indirect restorations after gingival maturation and pulpal maturity, or delay extractions of compromised teeth until an optimal age. The 'Hall Technique', which involves no tooth preparation, has demonstrated favourable results.[26] It offers the advantage of limited pulpal irritation, it is quick to perform, no local anaesthetic is required and caries is not removed. However, to date, studies have largely assessed the effectiveness of this technique for primary molars. These prefabricated crowns are also associated with gingival inflammation and therefore, when the time comes to replace them with definitive restorations, impression taking may need to be delayed until suitable gingival health is achieved.

The first molars should be assessed early for restorability and, where they are deemed of poor long-term prognosis, extraction with or without orthodontic treatment should be considered. This should ideally be done when the bifurcation of the second molars begins to calcify (usually age 8½–9½) to promote their more mesial eruption.[4] When extractions are required for orthodontic alignment, extraction of poor prognosis molars is preferred over extraction of unaffected teeth.

Lithium disilicate restorations were provided in Cases 1 and 2, which have shown favourable success rates for both posterior and anterior teeth.[27–29] In Case 2, ceramic crowns provided a more aesthetic and conservative alternative to the previous metal ceramic restorations. A systematic review expressed concerns that the evidence to support posterior lithium disilicate restorations is limited,[30] however, a more recent review demonstrated comparable success rates to metal ceramic alternatives.[31] All-ceramic lithium disilicate crowns may be manufactured via heat pressing or CAD/CAM techniques. Anadioti *et al* found that conventional impressions and heat pressing still provides the greatest marginal fit over CAD/CAM manufacture.[32] However, digital technology is changing rapidly and many dental laboratories have invested in CAD/CAM manufacture of all-ceramic restorations. In terms of survival, both CAD/CAM and pressed techniques have shown no difference in success over 7 years.[33]

Incisors

Patients may also show concerns about appearance, particularly if incisor teeth are affected. Enamel defects affecting incisors tend to be milder than the molars. However, treatment is often required due to aesthetic implications. Minimally invasive techniques, such as bleaching, microabrasion and resin infiltration may partially eliminate incisor lesions to varying degrees.[34,35] Microabrasion and resin infiltration work effectively on superficial lesions.[36] Microabrasion has been found to work more effectively on brown discoloration over white lesions.[36] A full description of microabrasion techniques is beyond the scope of this article, however, the authors would recommend techniques covered by Wallace and Deery.[36]

For bleaching, a 'walking technique' is recommended, with 10% carbamide peroxide (or 6% hydrogen peroxide) within a bleaching tray with incorporated buccal reservoirs.[36] This bleaching tray should be placed after toothbrushing and interdental cleaning to be worn overnight.[37] The desired affects are usually reached within 2–6 weeks.[37] Bleaching may be contra-indicated if the incisors have existing sensitivity as this may be exacerbated. The use of desensitizing agents will allow most patients to tolerate the bleaching process, however, should profound sensitivity occur, the bleaching process should be halted. Initial regular review is recommended to ensure that the patient is not experiencing acute sensitivity and that the bleaching is effective on the enamel lesions.

Direct composite is often required to mask the enamel defects, which provides a conservative and predictable outcome (Figures 2 and 4). Opaque composites can be used in conjunction with regular shades to mask dark brown hypoplasia. Traditionally, indirect restorations are more destructive and there are significant implications on pulpal vitality,[38] with up to 30% to 72% tooth tissue removal for veneers and full coverage crowns, respectively.[39] If direct restorations are carried out at a young age, direct composite addition and further polishing can be performed after gingival maturation. As gingival maturation is hard to predict, indirect restorations can leave unaesthetic margins, which can be challenging to manage and often require replacement.

Summary

In cases of enamel defects it may be challenging to identify the aetiology and reach a definitive diagnosis. However, the treatment strategies are often similar and depend largely on the severity of the lesions and not the aetiology. Where the diagnosis is MIH, it is important to discuss the long- and short-term implications of this with parents and patients. Patients are more prone to caries and pain in affected teeth and there may be aesthetic concerns.

Although there are often a number of restorative options available, considering the majority of patients present at a young age, conservative methods of intervention should be given priority. However, where the teeth are severely compromised, indirect restorations continue to have an important role. The cases presented demonstrate the use of a combination of indirect and direct techniques with excellent aesthetic and functional outcomes.

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