Molar Incisor Hypomineralization: Review and Recommendations for Clinical Management

Vanessa William, BDSc, DClinDent[1] Louise B Messer, BDSc, LDS, MDSc, PhD[2]
Michael F Burrow, BDSc, MDS, PhD, MEd[3]

Abstract

Molar incisor hypomineralization (MIH) describes the clinical picture of hypomineralization of systemic origin affecting one or more first permanent molars (FPMs) that are associated frequently with affected incisors. Etiological associations with systemic conditions or environmental insults during the child's first 3 years have been implicated. The complex care involved in treating affected children must address their behavior and anxiety, aiming to provide a durable restoration under pain-free conditions. The challenges include adequate anaesthesia, suitable cavity design, and choice of restorative materials. Restorations in hypomineralized molars appear to fail frequently; there is little evidence-based literature to facilitate clinical decisions on cavity design and material choice. A 6-step approach to management is described: (1) risk identification; (2) early diagnosis; (3) remineralization and desensitization; (4) prevention of caries and posteruption breakdown; (5) restorations and extractions; and (6) maintenance. The high prevalence of MIH indicates the need for research to clarify etiological factors and improve the durability of restorations in affected teeth. The purpose of this paper was to describe the diagnosis, prevalence, putative etiological factors, and features of hypomineralized enamel in molar incisor hypomineralization and to present a sequential approach to management. (Pediatr Dent 2006;28:224-232)

KEYWORDS: ENAMEL HYPOMINERALIZATION, MOLAR , DENTAL HYPOMINERALIZATION, ENAMEL OPACITY

Received July 8, 2005 Revision Accepted January 18, 2006

The term molar incisor hypomineralization (MIH) was introduced in 2001 to describe the clinical appearance of enamel hypomineralization of systemic origin affecting one or more permanent first molars (PFMs) that are associated frequently with affected incisors.[1] Also referred to as "hypomineralized" PFMs,[2] "idiopathic enamel hypomineralization,"[3,4] "dysmineralized" PFMs,[5] "nonfluoride hypomineralization,"[6,7] and "cheese molars,"[8,9] the condition is attributed to disrupted ameloblastic function during the transitional and maturational stages of amelogenesis.[3,10]

MIH's clinical management is challenging due to:

1. the sensitivity and rapid development of dental caries in affected PFMs;
2. the limited cooperation of a young child;
3. difficulty in achieving anesthesia; and
4. the repeated marginal breakdown of restorations.

Research on adhesion of restorative materials to hypomineralized enamel is limited, and clinical decisions to date have not been evidence-based. This may reflect a paucity of extracted hypomineralized molars with suitable surfaces for in vitro studies. A recently developed microshear bond strength test has allowed initial studies of the bond strength of materials to small surface areas of hypomineralized enamel.[11]

The purpose of this paper was to describe the diagnosis, prevalence, putative etiological factors, and features of hypomineralized enamel in molar incisor hypomineralization and to present a sequential approach to management.

MIH diagnosis

Criteria for the diagnosis of demarcated opacities, posteruption breakdown (PEB), atypical restorations, and extracted PFMs due to MIH were developed by Weerheijm et al.[12] Dentitions with generalized opacities present on all teeth (such as in several forms of amelogenesis imperfecta), rather than limited to the PFMs and permanent incisors, are not considered to have MIH.[12] After thorough cleaning, the

[1]Dr. William is honorary dental officer at the Royal Children's Hospital, Melbourne, Australia, and was a former postgraduate student in Pediatric Dentistry, [2]Dr. Messer is the Elsdon Storey Professor of Child Dental Health and Director of Graduate Studies, and [3]Dr Burrow is associate professor in Restorative Dentistry and Clinical Dean, all at the School of Dental Science, University of Melbourne, Victoria, Australia.
Correspond with Dr. Messer at ljbm@unimelb.edu.au

4 PFMs and 8 erupted permanent incisors are examined wet for demarcated opacities, PEB, and atypical restorations.[12] Demarcated opacities are defects of altered enamel translucency; the defective enamel is white-cream or yellow-brown in color, of normal thickness with a smooth surface, and has a distinct boundary adjacent to normal enamel.[2,13]

The opacities are usually limited to the incisal or cuspal one third of the crown, rarely involving the cervical one third.[2] The intact enamel surface is typically hard, smooth, and often hypermineralized following post-eruptive maturation; the subsurface enamel is soft and porous.[2] Having unusual size and shape, restorations may not conform to typical caries patterns and frequently involve the cuspal or incisal one third of the crown.[12] Enamel opacities may occur adjacent to restoration margins. Where one or more PFMs have been extracted, teeth in an otherwise sound dentition should be examined for possible causes (eg, opacities) and the child's history should be assessed for putative etiological factors.

Teeth with developmental defects of enamel may present similarly, regardless of etiology, and the development defects of enamel hypoplasia may be confused with MIH. Enamel hypoplasia (EH) is a quantitative defect associated with a reduced localized thickness of enamel, following disruption of the secretory phase of amelogenesis. The enamel may be translucent or opaque, with single or multiple pits or grooves and partial or complete absence of enamel over significant areas of dentin.[2,13] The EH defects tend to occur in the incisal or cuspal one third of the crown. Diagnostically, MIH and EH can be difficult to differentiate when affected molars have PEB due to caries or masticatory trauma. In a child with a high caries rate, MIH can be masked by extensive caries or restorations. Also, EH and MIH can occur together, particularly at a histological level.[13,14]

Hypomineralized enamel characteristics

Enamel is a highly mineralized tissue of ectodermal origin, secreted from ameloblasts that differentiate from the internal dental epithelium. Hypomineralization is thought to follow deposition of the full thickness of enamel matrix. The transitional ameloblast is considered most vulnerable. When these cells do not undergo complete maturation, full-thickness hypomineralization occurs.[3] Enamel maturation involves:

1. the removal of acid-labile mineral;
2. replacement with more acid-resistant apatite; and
3. an influx of calcium and phosphate ions, increasing the crystal width and thickness.[15]

Hypomineralization is thought to be due to disturbed resorptive potential of ameloblasts and proteolytic enzyme inhibition, leading to protein retention (particularly amelogenin) and interference with crystal growth and enamel maturation.[10,14,16,17] Regulation of pH during mineralization is considered necessary for normal apatite deposition and crystallite growth.[18] Investigating the relationship between enamel matrix pH during the maturation phase in cystic fibrosis mice, Sui et al[18] reported that reduced enamel matrix pH disrupted crystal growth and proteinase function. This resulted in protein retention and hypomineralization. This supported rat studies where hypomineralization followed altered pH after respiratory acidosis.[19] Speculatively, such conditions affecting matrix pH during enamel maturation may predispose MIH.[18]

Lack of calcium phosphate may also contribute to the formation of hypomineralized enamel.[8] Using secondary ion mass spectrometry and X-ray microanalysis, increasing severity of hypomineralization correlated positively with increasing carbon concentration and decreasing concentrations of calcium and phosphorus. This resulted in significantly lowered calcium/phosphorous ratios in the enamel.[20] This also supported other studies where cystic fibrosis transmembrane regulator knockout mice had decreased levels of calcium and calcium/phosphorus ratios in hypomineralized enamel when compared to normal enamel.[21] Thus, impaired calcium metabolism may have a role in the development of hypomineralized enamel.[20,21]

The color of hypomineralized enamel defects may reflect differences in hardness, porosity, and mineral content. Yellow-brown defects have lower Knoop hardness values and greater porosity than white defects and normal enamel.[2,22] Nano-indentation studies have shown significantly lower values for hardness and modulus of elasticity than seen in to unaffected enamel.[23] Under scanning electron microscopy, the defects showed increased porosity and disorganized rod structure of fractured surfaces.[23]

Clinically, PFMs can differ in defect severity in an individual where all 4 molar germs appear to have been subjected to the same systemic disorder.[3,24] Concurrently mineralizing PFMs may show randomly distributed opacities in single or several molars in the same individual.[2,3] The gradient of mineral content of these opacities decreases from the dentoenamel junction to the subsurface enamel (the reverse of normal enamel); usually the surface layer becomes hypermineralized with post-eruptive maturation.[3] Zones of apparently sound enamel in hypomineralized molars show an overall reduction in mineral concentration (of about 5%) and lower calcium/phosphorus ratios, indicating that the entire crown is affected to some extent.[3,20] Clinical examples of affected PFMs are shown in Figures 1a to 1d.

Putative factors associated with molar incisor hypomineralization

Putative factors associated with disrupted amelogenesis of PFMs include systemic conditions and environmental insults influencing natal and early development.[8,25,26] A recent study in Greece of 151 MIH children reported that 78% had experienced medical problems: (1) prenatally (19%); (2) perinatally (44%); and (3) neonatally (22%).[27] Only 15% of the children did not appear to have a putative etiological factor in their history.[27]

Causal relationships, however, cannot be assigned definitively from studies relying on parental recall of medical and dental events in their child's first 3 years.[1,3] Determina-

tion of etiological factors is also complicated when a child has more than one medical problem in this time period. Although a number of etiological factors may contribute to MIH, the threshold level needed to cause enamel defects at sensitive stages of amelogenesis is unknown.[28] While systemic illnesses may not produce a developmental defect of enamel when experienced singly, 2 or more concurrent conditions may act synergistically to produce a defect.[2,28] This is illustrated in a study of 53 Swedish children with 22q11 microdeletion syndrome.[29] Phenotypically, this multiple anomaly has a characteristic facies, and may include congenital heart defects, velopharyngeal insufficiency with or without cleft palate, immune problems, feeding difficulties, hypocalcemia, learning disabilities, behavioural problems, and skeletal, neurologic, and gastrointestinal abnormalities.[29] Of 47 affected children, 3 (6%) had EH in the permanent dentition and 16 (34%) had hypomineralized permanent teeth. Computerized inductive analyses showed that the EH of permanent and primary teeth correlated with prematurity and heart defects (30%) and enamel hypomineralization correlated with frequent preschool age infections and heart defects (43%).[29]

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.[2,8,26] In a retrospective study of 21 Dutch MIH children, 67% had suffered from bronchitis, asthmatic bronchitis, pneumonia, and upper respiratory tract infections.[8] Antibiotic usage has also been implicated. Due to the concurrence of disease and antibiotic therapy, however, it is difficult to ascertain whether the MIH was associated with the disease or the antibiotic.[26] Children with poor general health and systemic conditions are more likely to have developmental defects of enamel.[30,31] The systemic conditions implicated to date include nutritional deficiencies, brain injury and neurologic defects, cystic fibrosis, syndromes of epilepsy and dementia (Kohlschutter-Tonz syndrome), nephrotic syndrome, atopia, lead poisoning, repaired cleft lip and palate, radiation treatment, rubella embryopathy, epidermolysis bullosa, ophthalmic conditions, celiac disease, and gastrointestinal disorders.[30,32-34]

Preterm birth has been associated with increased prevalence of enamel defects, including hypomineralization and hypoplasia in the permanent dentition.[33,35-37] As the PFM commences mineralization soon after birth, a persistent systemic derangement postnatally may affect enamel mineralization.[35] Preterm birth can be associated with respiratory difficulties, hyperbilirubinaemia, metabolic disturbances including hypocalcemia and hypoglycemia, haematological disorders, patent ductus arteriosus, and intracranial hemorrhage.[38] A study of 32 Finnish children 9 to 11 years old found enamel defects in 36% of children born fullterm and 84% of children born preterm.[36] A study of 40 children born preterm with very low birthweight (<1,500 gm) in Brisbane, Australia showed a significantly higher percentage of enamel defects in their PFMs (17%) than in a matched sample of normal birthweight children (8%).[35] The enamel defect severity increased with decreasing gestational age and lower birthweight.[35]

Associations have been made between the presence of polychlorinated dibenzo-*p*-dioxins (PCDDs) in breast milk and enamel hypomineralization in both clinical and laboratory studies.[39-41] The PCDDs belong to a class of environmental pollutants known as polyhalogenated aromatic hydrocarbons.[39] Persistence and accumulation of PCDDs in tissue lipids and in the food chain may result in chronic low-level exposure in humans.[42] In studies of Finnish children examined for hypomineralization of PFMs, increases in severity and numbers of defects were seen in those exposed to higher amounts of PCDD and furan via their mother's breast milk compared to those less exposed.[39,40]

Although other clinical studies have not found associations between dioxin compounds in breast milk and

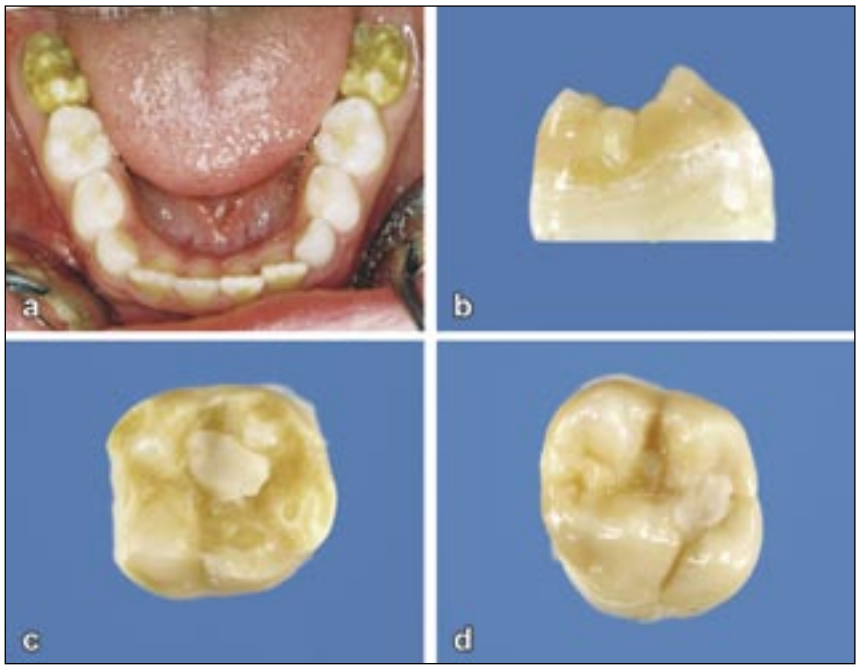

Figure 1a. Clinical case of right and left permanent first molars severely affected by molar incisor hypomineralization.
Figure 1b. Permanent first molar showing yellow-brown demarcated opacity of enamel.
Figure 1c: Mandibular permanent first molar affected by molar incisor hypomineralization, extensive post-eruptive breakdown, and marginal breakdown surrounding a restoration.
Figure 1d. Maxillary permanent first molar affected by molar incisor hypomineralization, extensive post-eruptive breakdown, and marginal breakdown surrounding a restoration.

Table 1. Recent Studies Reporting Prevalence of Molar Incisor Hypomineralization (MIH)

Study and year of publication	Study sample	First permanent molar (PFM) prevalences reported
Koch et al (1987)[4]	2,226 Swedish children born 1966-1974	4%-15% children had "idiopathic hypomineralization" of PFMs
Alaluusua et al (1996)[39]	102 Finnish children 6-7 ys old	17% of children had hypomineralized PFMs
Alaluusua et al (1996)[40]	97 Finnish children 12 ys old with a history of extensive and prolonged breast-feeding	25% children had PFM hypomineralization
Jalevik et al (2001)[43]	516 Swedish children 7-8 ys old	18% children had MIH; affected children averaged 3.2 hypomineralized teeth, of which 2.4 were PFMs
Leppaniemi et al (2001)[7]	488 Finnish children 7-13 ys old	19% of children had "nonfluoride hypomineralizations" of PFMs
Weerheijm et al (2001)[9]	497 Dutch children 11 ys old and born in 1988	10% of children had "cheese molars (idiopathic enamel disturbances)"; 79% of affected children had 2 or more affected PFMs
Dietrich et al (2003)[45]	2,408 German children 10-17 ys old and born 1985-1992	6% of children had MIH; affected children averaged 4.8 hypomineralized teeth, of which 2.2 were PFMs
Lygidakis et al (2004)[27]	2,640 Greek children attending a children's community health center	6% children had MIH
Kosem et al (2004)[44]	2,339 Slovenian adolescents 12-18 ys old	14% of children had at least 1 PFM with "demarcated opacity"
Chawla et al (2004)[46]	Australian children attending a large pediatric dental specialist referral practice	Of 182 MIH children, 70% had ≥1 affected PFMs

hypomineralized enamel,[6,26] a positive association was demonstrated when 2, 3, 7, 8-tetrachlorodibenzo-*p*-dioxin was administered to lactating rats. The arrested degradation and removal of matrix proteins resulted in protein retention and hypomineralized enamel.[42] Future studies should elucidate if children exposed to such environmental pollutants may be at risk for MIH. Although the etiology may be unclear, children with poor general health in the first 3 years who were born preterm or who were exposed to certain environmental contaminants may be at risk for MIH.

Table 2. A Clinical Management Approach for Permanent First Molars Affected by Molar Incisor Hypomineralization

Steps	Recommended procedures
Risk identification	Assess medical history for putative etiological factors
Early diagnosis	Examine at-risk molars on radiographs if available
	Monitor these teeth during eruption
Remineralization and desensitization	Apply localized topical fluoride
Prevention of dental caries and post-eruption breakdown (PEB)	Institute thorough oral hygiene home care program
	Reduce cariogenicity and erosivity of diet
	Place pit and fissure sealants
Restorations or extractions	Place intracoronal (resin composite) bonded with a self-etching primer adhesive or extracoronal restorations (stainless steel crowns)
	Consider orthodontic outcomes post-extraction
Maintenance	Monitor margins of restorations for PEB
	Consider full coronal coverage restorations in the long term

Prevalence of molar incisor hypomineralization

The limited prevalence data for MIH reflects several diagnostic classifications. Using the criteria of Weerheijm et al,[12] the prevalence ranges from 4% to 25%.[24] Recent studies reporting prevalences from several countries are shown in Table 1. The number of hypomineralized PFMs in an individual can vary from 1 to 4, affecting particularly 2 or more molars including the contralateral tooth, where the teeth are moderately or severely affected.[9,39,43-46] The risk of involvement of the permanent maxillary incisors appears to increase when more PFMs are affected.[1,43,47]

Risk identification, remineralization, and preventive management

MIH children often experience PFM pain and sensitivity and aesthetic concerns when their incisors are affected. A 6-step management approach is proposed (Table 2). Children at risk for MIH should be identified prior to PFM eruption, based upon a relevant history of putative etiological factors in the first 3 years and from careful study under magnification of the unerupted molar crowns on any available radiographs.

During PFM eruption, the hypomineralized surface is very susceptible to caries and erosion. The cariogenicity and erosivity of the child's diet should be assessed and appropriate recommendations made for dietary modifica-

tion. Thorough oral hygiene should be instituted; this could include a desensitizing toothpaste. Remineralization therapy should commence as soon as the defective surface is accessible, aiming to produce a hypermineralized surface layer and to desensitize the tooth.[3,48] Remineralization and desensitization may be accomplished with casein phosphopeptide-amorphous calcium phosphate (CPP-ACP) oral care products. The CPP-ACP can interact with fluoride ions, producing an amorphous calcium phosphate stabilized by CPP at the tooth surface and providing soluble calcium, fluoride, and phosphate ions to promote remineralization with fluorapatite that is more acid resistant.[49] Three CPP-ACP oral care products are commercially available in Australia, the United States, Europe, and Japan:

1. a topical tooth crème (Tooth Mousse or MI Paste, GC Corporation, Tokyo, Japan);
2. a sugar-free chewing gum (Recaldent Pty Ltd, Melbourne, Australia); and
3. lozenges (Adams/Cadbury Schweppes, Morris Plains, NJ) containing CPP-ACP as Recaldent (Recaldent Pty Ltd, Melbourne, Australia).[49]

These products enhance remineralization by creating a state of supersaturation followed by deposition of calcium and phosphate ions at the enamel surface.[49] While clinical protocols for CPP-ACP oral care products await development, anecdotal reports describe surface hardening and reduction in tooth sensitivity from daily home use.[50]

Topical fluoride, delivered as concentrated varnishes or gels, can remineralize enamel, reduce sensitivity, and enhance resistance to demineralization by providing a reservoir of fluoride ions for redeposition as fluorapatite during remineralization.[51] Anecdotal reports ascribe considerable clinical benefit to topical application of fluoride on hypomineralized molars, resulting in surface hardening of demineralized enamel prior to restoration.[48] One such varnish (Duraphat, Colgate Oral Care, Sydney, Australia) containing 50 mg NaF/mL (2.26% F, 22,600 ppm F) binds to enamel and plaque, acting as a slow-release fluoride reservoir. A less concentrated treatment gel (Gelkam, Colgate Oral Care, Sydney, Australia) containing 0.4% SnF (3,000 ppm Sn and 1,000 ppm F) can be applied as a single drop on a cotton bud by the parent (told to "paint on the molar like nail polish") several times per week after brushing and flossing. The parent must be very responsible and fully compliant in undertaking this task, as regular swallowing of this product prior to eruption of the permanent incisors could promote anterior fluorosis.[51]

In summary, the oral hygiene strategies that could be given to parents or patients in cases where tooth-brushing is difficult due to sensitive, poorly mineralized surfaces of affected molars are as follows:

1. brush affected molars gently with a desensitizing toothpaste (preferably containing fluoride) on a soft toothbrush;
2. apply a CPP-ACP topical crème daily using a cotton bud; and
3. apply a low concentration fluoride treatment gel regularly using a cotton bud.

As remineralization and desensitization of the affected molars occurs, regular oral hygiene strategies can be instituted.

For partially erupted PFMs where moisture control is suboptimal, glass ionomer cement sealants can provide caries protection and reduce surface permeability. Retention is poor, however, and such sealants may need rebuilding later with a resin-based sealant when optimal moisture control is possible.[52,53] Without preventive care, hypomineralized PFMs are at risk of PEB in the acidic and masticatory challenges of the oral cavity. When PEB occurs, the porous subsurface enamel or dentin is exposed, resulting in teeth sensitive to cold air, warm water, and tooth-brushing.[1] Poor oral hygiene favors plaque retention and promotes rapid caries development.[7,28,54]

Restoring hypomineralized first permanent molars

Restoring affected PFMs is complicated frequently by:

1. difficulties in achieving anesthesia;
2. managing the child's behaviour;
3. determining how much affected enamel to remove; and
4. selecting a suitable restorative material.[1]

The porous exposed subsurface enamel or dentin may promote chronic inflammation of the pulp, complicating anaesthesia.[48,55,56] The adjunctive use of nitrous oxide-oxygen analgesia may alleviate anxiety and reduce dental pain, or general anesthesia may be required for restorative treatment. In determining cavity margin placement, 2 approaches are described:

1. All defective enamel is removed.
2. Only the very porous enamel is removed, until good resistance of the bur to enamel is felt.[48]

The former approach may avoid premature restoration failure, but sacrifices tooth structure; the latter approach is conservative, but places restorations at risk of marginal breakdown. Removal of all defective enamel is recommended when bonding resin composite restorations to hypomineralized PFMs due to the poor bond strength of resin adhesives to hypomineralized enamel.[57]

MIH children receive much more dental treatment than unaffected children.[7,55,58] A retrospective Swedish study of 32 MIH children and 41 control children showed that by age 9, those with MIH had undergone treatment of their PFMs nearly 10 times more frequently than control children; on average, each defective molar had been treated twice due to restoration failure, PEB, or recurrent caries.[55] In the control group, no molars were retreated and local anaesthesia was often unnecessary.[55] Consequently, it is not surprising that a MIH child who has had pain, difficulties with anaesthesia, and retreatment develops poor behaviour and dental anxiety.[55] The complex care involved in treating such a child must address their behaviour and anxiety, aiming to provide a durable restoration under pain-free conditions.

The choice of materials will depend on the defect severity and the age and cooperation of the child.[48,54] Restorative options include glass ionomer cements (GIC), resin-modified glass ionomer cements (RMGIC), polyacid modified resin composites (PMRC), resin composites (RC), amalgam, stainless steel crowns (SSCs), and indirect adhesive or cast onlays or crowns. Amalgam is the least durable due to:

1. poor retention in shallow cavity preparations; and
2. the inability to protect remaining tooth structure, which is likely to result in restoration failure.[48]

Adhesive materials are usually chosen due to the atypical cavity outlines following removal of hypomineralized enamel.[54] For dentin replacement or as an interim restoration, GIC provides: (1) placement ease; (2) fluoride release; and (3) chemical bonding.[54,56,59] The RMGICs offer similar advantages to GIC; the incorporation of resin and photoinitiators improves: (1) handling; (2) wear resistance; (3) fracture toughness; and (4) fracture resistance.[60] Restorations of GIC or RMGIC are not recommended in stress-bearing areas, such as occlusal surfaces of hypomineralized molars, but may suffice until a definitive restoration is achievable.[54,56,59,60]

With physical properties superior to GIC and RMGIC, the RCs are esthetic materials with high wear resistance and adhesion when used with resin-based adhesives; they can be used solely or in a sandwich technique following previous temporization with GIC. RCs are technique sensitive, however, requiring good moisture control under rubber dam and long placement time.[61] The RCs are materials of choice in MIH where defective enamel is well demarcated and confined to 1 or 2 surfaces with supragingival margins and without cuspal involvement.[48] Restoring affected PFMs with RC was clinically successful over 4 years when all defective enamel was removed.[47] The PMRCs:

1. have good handling characteristics;
2. release and take up fluoride; and
3. have tensile and flexural strength properties superior to GIC and RMGIC, but inferior to that of RC.[62]

The use of PMRCs in permanent teeth is restricted to nonstress-bearing areas with limited application in hypomineralized PFMs.[61,63]

Restoring hypomineralized permanent incisors

Hypomineralized incisors in MIH may present esthetic concerns to children and their parents.[10,48] Microabrasion can be an effective treatment in shallow defects, but the defects usually extend through the full enamel thickness.[48] A conservative approach in managing yellow-brown hypomineralized enamel involves:

1. etching the lesion with 37% phosphoric acid;
2. bleaching with 5% sodium hypochlorite; and then
3. re-etching the enamel prior to placing a sealant over the surface to occlude porosities and prevent restaining.[64]

One report of this approach described clinical success with little or no staining up to 5 years post-treatment.[64] Others report little improvement with acid/pumice microabrasion used alone, but esthetic improvement was achieved when the enamel reduction was combined with opaque resins then direct RC veneering.[24,48,65] Porcelain veneers are typically delayed until late adolescence when the teeth have fully erupted and the gingival architecture has stabilized.[64]

Adhesion to hypomineralized enamel

The limited literature on adhesion of dental materials to hypomineralized enamel has focused on case reports of amelogenesis imperfecta (AI). Hypomineralization appears most severe in the hypocalcified and hypomaturation types of AI, where the decreased mineral content and increased protein content of the enamel limit effective etching and bonding.[64,66,67] Enamel pretreatment with 5% sodium hypochlorite to remove protein encasing the hydroxyapatite has been suggested.[64,68] Removal of all hypomineralized enamel prior to placing RC restorations has been recommended.[47,57,69-71] Literature on bonding to hypomineralized enamel is limited, perhaps reflecting the paucity of extracted hypomineralized teeth with suitable surfaces for bond strength testing.

Bond strengths of RC to hypomineralized enamel of PFMs affected with MIH are significantly less than bond strengths to normal enamel for both single-bottle total etch and self-etching primer adhesives.[57] A recent laboratory study by the present authors showed the mean microshear bond strengths (MPa) of resin composite bonded to hypomineralized enamel was significantly lower than for control enamel (3M ESPE Single Bond, St Paul, Minn: 7.08±4.90 vs 16.27±10.04; Clearfil SE Bond, Kuraray Medical Inc, Tokyo, Japan: 10.39±7.56 vs 19.63±7.42; P=.001).[57] After phosphoric acid etching, scanning electron microscopy of hypomineralized enamel showed interprismatic spaces and very little intercrystal porosity within the enamel prisms, allowing limited microtag formation (important for bonding effectiveness of single-bottle, total-etch adhesive systems) and weaknesses that could lead to crack propagation within the enamel.[57] In restoring molar surfaces with limited involvement, RC is recommended. After removing all discolored hypomineralized enamel, cavity margins should be placed on apparently sound enamel and RC should be bonded with a self-etching primer adhesive. Marginal placement should be located on apparently sound enamel due to the poor adhesion of RC to hypomineralized enamel.[57]

Full coronal coverage restorations

When PFMs have moderate to severe PEB, preformed SSCs are the treatment of choice.[48,54,56,72] These crowns:

1. prevent further tooth deterioration;
2. control tooth sensitivity;
3. establish correct interproximal contacts and proper occlusal relationships;
4. are not as technique sensitive or costly as cast restorations; and
5. require little time to prepare and insert.[54,73,74]

If not adapted properly, however, SSCs may produce an open bite, gingivitis, or both.[75] Properly placed, SSCs can preserve PFMs with MIH until cast restorations are feasible.[71,72]

Partial and full coverage indirect adhesive or cast crowns and onlays may be considered for MIH in the late mixed and permanent dentitions.[54,71,76] Such restorations are rarely indicated for PFMs in young children due to placement difficulties associated with: (1) short crowns; (2) large pulps; (3) long treatment time and high cost; and (4) the child's limited cooperation.[48,75] The use of laboratory-fabricated crowns of cast gold, indirect composite, and ceramic placed in 6- to 8-year-old children, however, has been described as clinically very successful over a 2- to 5-year follow-up.[75] Compared to SSCs, cast restorations:

1. require minimal tooth reduction;
2. minimize pulpal trauma;
3. protect tooth structure;
4. provide high strength for cuspal overlays;
5. control sensitivity; and
6. maintain periodontal health due to their supragingival margins.[71,76]

Others argue no difference in quality or longevity between cast adhesive copings and preformed SSCs.[73] Therefore, the decision to restore hypomineralized PFMs with either cast adhesive copings or preformed SSCs is based on:

1. the patient's immediate and long-term needs;
2. their cooperation;
3. treatment cost; and
4. the clinician's skills and material choices.

Extraction of severely hypomineralized first permanent molars

When PFMs are severely hypomineralized, restorations may be impossible and extraction must be considered.[9,55] In such cases, early orthodontic assessment is recommended. Since PFMs are rarely an orthodontist's choice for extraction, later orthodontic treatment may be complicated.[77] Factors affecting molar prognosis—such as vitality and restorability, dental age, buccal segment crowding, occlusal relationships, and the condition of other erupted and unerupted teeth—all need to be assessed when considering molar extraction.[48,78] If restorative treatment is a major problem, or if it fails, the optimal timing of extractions and follow-up of tooth eruption and development of occlusion can be managed.

Conclusions

The prevalence of MIH appears to be increasing, and managing affected children is now a common problem for pediatric dentists. Although the etiology is unclear and may, in fact, be multifactorial, children born preterm and those with poor general health or systemic conditions in their first 3 years may develop MIH. The early identification of such children will allow monitoring of their PFMs so that remineralization and preventive measures can be instituted as soon as affected surfaces are accessible. The complex care involved must address the child's behaviour and anxiety, aiming to provide durable restorations under pain-free conditions. Restoration of surfaces with limited involvement with resin composite is recommended following:

1. removal of all discolored hypomineralized enamel;
2. placement of cavity margins on apparently normal enamel; and
3. bonding with a self-etching primer adhesive.

Extensively affected molars may require extracoronal restorations or extraction. Research is needed to clarify etiological factors and improve the durability of restorations in affected teeth.

References

1. Weerheijm KL, Jalevik B, Alaluusua S. Molar-incisor hypomineralization. Caries Res 2001;35:390-391.
2. Jalevik B, Noren JG. Enamel hypomineralization of permanent first molars: A morphological study and survey of possible aetiological factors. Int J Paediatr Dent 2000;10:278-289.
3. Fearne J, Anderson P, Davis GR. 3D X-ray microscopic study of the extent of variations in enamel density in first permanent molars with idiopathic enamel hypomineralization. Br Dent J 2004;196:634-638.
4. Koch G, Hallonsten AL, Ludvigsson N, Hansson BO, Holst A, Ullbro C. Epidemiologic study of idiopathic enamel hypomineralization in permanent teeth of Swedish children. Community Dent Oral Epidimiol 1987;15:279-285.
5. Croll TP. Creating the appearance of white enamel dysmineralization with bonded resins. J Esthet Dent 1991;3:30-33.
6. Holtta P, Kiviranta H, Leppaniemi A, Vartiainen T, Lukinmaa PL, Alaluusua S. Developmental dental defects in children who reside by a river polluted by dioxins and furans. Arch Environ Health 2001;56:522-528.
7. Leppaniemi A, Lukinmaa PL, Alaluusua S. Nonfluoride hypomineralizations in the permanent first molars and their impact on the treatment need. Caries Res 2001;35:36-40.
8. van Amerongen WE, Kreulen CM. Cheese molars: A pilot study of the etiology of hypocalcifications in first permanent molars. J Dent Child 1995;62:266-269.
9. Weerheijm KL, Groen HJ, Beentjes VE, Poorterman JH. Prevalence of cheese molars in 11-year-old Dutch children. J Dent Child 2001;68:259-262.
10. Wright JT, Hall K, Yamauchi M. The protein composition of normal and developmentally defective enamel. Ciba Found Symp 1997;205:85-99, 106 (discussion).
11. McDonough WG, Antonucci JM, He J, et al. A microshear test to measure bond strengths of dentin-polymer interfaces. Biomaterials 2002;23:3603-3608.
12. Weerheijm KL, Duggal M, Mejare I, Papagiannoulis L, Koch G, Martens LC, et al. Judgement criteria for molar incisor hypomineralization (MIH) in epidemiologic studies: a summary of the European meeting on MIH held in Athens, 2003. Eur J Paediatr Dent 2003;4:110-113.

13. Commission on Oral Health Research & Epidemiology. A review of the developmental defects of enamel index (DDE Index). Commission on Oral Health, Research & Epidemiology. Report of an FDI Working Group. Int Dent J 1992;42:411-426.
14. Suga S. Enamel hypomineralization viewed from the pattern of progressive mineralization of human and monkey developing enamel. Adv Dent Res 1989;3:188-198.
15. Avery J. *Oral Development and Histology*. 3rd ed. Stuttgart, Germany: Thieme; 2002.
16. Sato K, Hattori M, Aoba T. Disturbed enamel mineralization in a rat incisor model. Adv Dent Res 1996;10:216-224.
17. Robinson C, Brookes SJ, Bonass WA, Shore RC. Enamel maturation. Ciba Found Symp 1997;205:156-174.
18. Sui W, Boyd C, Wright JT. Altered pH regulation during enamel development in the cystic fibrosis mouse incisor. J Dent Res 2003;82:388-392.
19. Whitford GM, Angmar-Mansson B. Fluorosis-like effects of acidosis, but not NH_4^+ on rat incisor enamel. Caries Res 1995;29:20-25.
20. Jalevik B, Odelius H, Dietz W, Noren JG. Secondary ion mass spectrometry and X-ray microanalysis of hypomineralized enamel in human permanent first molars. Arch Oral Biol 2001;46**:**239-247.
21. Arquitt CK, Boyd C, Wright JT. Cystic fibrosis transmembrane regulator gene (CFTR) is associated with abnormal enamel formation. J Dent Res 2002;81:492-496.
22. Suckling GW, Nelson DG, Patel MJ. Macroscopic and scanning electron microscopic appearance and hardness values of developmental defects in human permanent tooth enamel. Adv Dent Res 1989;3:219-233.
23. Mahoney EK. Mechanical properties and microstructure of hypomineralized enamel of permanent teeth. Biomaterials 2004;25:5091-5100.
24. Weerheijm KL. Molar incisor hypomineralization (MIH): Clinical presentation, aetiology, and management. Dent Update 2004;31:9-12.
25. Brook AH, Fearne JM, Smith JM. Environmental causes of enamel defects. Ciba Found Symp 1997;205:21-25 (discussion), 212-221.
26. Jalevik B, Noren JG, Barregard L. Etiologic factors inflencing the prevalence of demarcated opacities in permanent first molars in a group of Swedish children. Eur J Oral Sci 2001;109:230-234.
27. Lygidakis NA, Dimou G, Marinou D, Gouva G. *Aetiology of Molar-incisor Hypomineralization.* A retrospective study of 151 children with the defect [abstract]. Barcelona, Spain: 7th Congress of the European Academy of Paediatric Dentistry; 2004.
28. Seow WK. Clinical diagnosis of enamel defects: Pitfalls and practical guidelines. Int Dent J 1997;47:173-182.
29. Klingberg G, Oskarsdottir S, Johannesson EL, Noren JG. Oral manifestations in 22q11 deletion syndrome. Int J Paediatr Dent 2002;12:14-23.
30. Hall R. The prevalence of developmental defects of tooth enamel (DDE) in a paediatric hospital department of dentistry population (part I). Adv Dent Res 1989;3:114-119.
31. Pascoe L, Seow WK. Enamel hypoplasia and dental caries in Australian Aboriginal children: Prevalence and correlation between the two diseases. Pediatr Dent 1994;16:193-199.
32. Seow WK. Enamel hypoplasia in the primary dentition: a review. J Dent Child 1991;58:441-452.
33. Martinez A, Cubillos P, Jimenez M, Brethauer U, Catalan P, Gonzalez U. Prevalence of developmental enamel defects in mentally retarded children. J Dent Child 2002;69:151-155.
34. Kirkham J, Robinson C, Strafford SM, Shore RC, Bonass WA, Brookes SJ, Wright JT. The chemical composition of tooth enamel in junctional epidermolysis bullosa. Arch Oral Biol 2000;45:377-386.
35. Seow WK. A study of the development of the permanent dentition in very low birthweight children. Pediatr Dent 1996;18:379-384.
36. Aine L, Backstrom MC, Maki R, Kuusela AL, Koivisto AM, Ikonen RS, Maki M. Enamel defects in primary and permanent teeth of children born prematurely. J Oral Pathol Oral Med 2000;29:403-409.
37. Seow WK. Effects of preterm birth on oral growth and development. Aust Dent J 1997;42:85-91.
38. Nguyen C. Problems of prematurity and its effect on growth and development. Synopses 1997;16:1, 6-11.
39. Alaluusua S, Lukinmaa PL, Vartiainen T, Partanen M, Torppa J, Tuomisto J. Polychlorinated dibenzo-p-dioxins and dibenzofurans via mother's milk may cause developmental defects in the child's teeth. Environ Toxicol Pharmacol 1996;1:193-197.
40. Alaluusua S, Lukinmaa PL, Koskimies M, Pirinen S, Holtta P, Kallio M, Holttinen T, Salmenpera L. Developmental dental defects associated with long breast-feeding. Eur J Oral Sci 1996;104:493-497.
41. Jan J, Vrbic V. Polychlorinated biphenyls cause developmental enamel defects in children. Caries Res 2000;34:469-473.
42. Gao Y, Sahlberg C, Kiukkonen A, Alaluusua S, Pohjanvirta R, Tuomisto J, Lukinmaa PL. Lactational exposure of Han/Wistar rats to 2, 3, 7, 8-tetrachlorobenzo-p-dioxin interferes with enamel maturation and retards dentin mineralization. J Dent Res 2004;83:139-144.
43. Jalevik B, Klingberg G, Barregard L, Noren JG. The prevalence of demarcated opacities in permanent first molars in a group of Swedish children. Acta Odontol Scand 2001;59:255-260.
44. Kosem R, Senk Erpic A, Kosir N, Kastelec D. *Prevalence of Enamel Defects With Emphasis on Molar Incisor Hypomineralization in Slovenian Children and Adolescents* [abstract]. Barcelona, Spain: 7th Congress of the European Academy of Paediatric Dentistry; 2004.

45. Dietrich G, Sperling S, Hetzer G. Molar incisor hypomineralization in a group of children and adolescents living in Dresden (Germany). Eur J Paediatr Dent 2003;4:133-137.
46. Chawla N, Messer LB, Silva M. Distribution and putative aetiological factors of molar-incisor hypomineralization and molar hypomineralization [abstract]. Aust Dent J 2004;49(suppl):S26-27.
47. Lygidakis NA, Chaliasou A, Siounas G. Evaluation of composite restorations in hypomineralized permanent molars: A four-year clinical study. Eur J Paediatr Dent 2003;4:143-148.
48. Fayle SA. Molar incisor hypomineralization: Restorative management. Eur J Paediatr Dent 2003;4:121-126.
49. Reynolds EC. New modalities for a new generation: Casein phosphopeptide-amorphous calcium phosphate, a new remineralization technology. Synopses 2005;30:1-6.
50. Kilpatrick N, Mahoney EK. Dental erosion: Part 2. The management of dental erosion. N Z Dent J 2004;100:42-47.
51. Messer LB. Getting the fluoride balance right: Children in long-term fluoridated communities. Synopses 2005;30:7-10.
52. Manton DJ, Messer LB. Pit and fissure sealants: Another major cornerstone in preventive dentistry. Aust Dent J 1995;40:22-29.
53. Simonsen RJ. Pit and fissure sealant: A review of the literature. Pediatr Dent 2002;24:393-414.
54. Mahoney EK. The treatment of localized hypoplastic and hypomineralized defects in first permanent molars. N Z Dent J 2001;97:101-105.
55. Jalevik B, Klingberg GA. Dental treatment, dental fear and behaviour management problems in children with severe enamel hypomineralization of their permanent first molars. Int J Paediatr Dent 2002;12:24-32.
56. Croll TP. Restorative options for malformed permanent molars in children. Compend Contin Educ Dent 2000;21:676-678, 680, 682.
57. William V. *Microshear Bond Strength of Resin Composite to Teeth Affected by Molar Incisor Hypomineralization* [DClinDent thesis]. Victoria, Australia: University of Melbourne; 2005.
58. Kotsanos N, Kaklamanos E, Arapostathis K. *Treatment Management of Permanent Molars in Children With Molar-incisor Hypomineralization* [abstract PO55]. Barcelona, Spain: 7th Congress of the European Academy of Paediatric Dentistry; 2004.
59. Berg JH. Glass ionomer cements. Pediatr Dent 2002;24:430-438.
60. Croll TP, Nicholson JW. Glass ionomer cements in pediatric dentistry: A review of the literature. Pediatr Dent 2002;24:423-429.
61. Donly K, Garcia-Godoy F. The use of resin-based composite in children. Pediatr Dent 2002;24:480-488.
62. Lyons K. Direct placement restorative materials for use in posterior teeth: The current opinions. N Z Dent J 2003;99:10-15.
63. Burgess JO, Walker R, Davidson JM. Posterior resin-based composite: Review of the literature. Pediatr Dent 2002;24:465-479.
64. Wright JT. The etch-bleach-seal technique for managing stained enamel defects in young permanent incisors. Pediatr Dent 2002;24:249-252.
65. Welbury RR. A clinical study of a microfilled composite resin for labial veneers. Int J Paediatr Dent 1991;1:9-15.
66. Wright JT, Deaton TG, Hall KI, Yamauchi M. The mineral and protein content of enamel in amelogenesis imperfecta. Connect Tissue Res 1995;32:247-252.
67. Takagi Y, Fujita H, Katano H, Shimokawa H, Kuroda T. Immunochemical and biochemical characteristics of enamel proteins in hypocalcified amelogenesis imperfecta. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85:424-430.
68. Venezie RD, Vadiakas G, Christensen JR, Wright JT. Enamel pretreatment with sodium hypochlorite to enhance bonding in hypocalcified amelogenesis imperfecta: Case report and SEM analysis. Pediatr Dent 1994;16:433-436.
69. Li RW. Adhesive solutions: Report of a case using multiple adhesive techniques in the management of enamel hypoplasia. Dent Update 1999;26:277-282, 284, 286-287.
70. Yip HK, Smales RJ. Oral rehabilitation of young adults with amelogenesis imperfecta. Int J Prosthod 2003;16:345-349.
71. Hunter L, Stone D. Supraoccluding cobalt-chrome onlays in the management of amelogenesis imperfecta in children: 12-year case report. Quintessence Int 1997;28:15-19.
72. Radcliffe RM, Cullen CL. Preservation of future options: restorative procedures on first permanent molars in children. J Dent Child 1991;58:104-108.
73. Zagdwon AM, Fayle SA, Pollard MA. A prospective clinical trial comparing preformed metal crowns and cast restorations for defective first permanent molars. Eur J Oral Sci 2003;3:138-142.
74. Randall RC. Preformed metal crowns for primary and permanent molar teeth: Review of the literature. Pediatr Dent 2002;24:489-500.
75. Koch MJ, Garcia-Godoy F. The clinical performance of laboratory-fabricated crowns placed on first permanent molars with developmental defects. J Am Dent Assoc 2000;131:1285-1290.
76. Harley KE, Ibbetson RJ. Dental anomalies: Are adhesive castings the solution? Br Dent J 1993;174:15-22.
77. Williams JK, Gowans AJ. Hypomineralized first permanent molars and the orthodontist. Eur J Paediatr Dent 2003;4:129-132.
78. Gill D, Lee R, Tredwin C. Treatment planning for the loss of first permanent molars. Dent Update 2001;28:304-308.