Hypomineralised second primary molars may be indicative of future molar incisor hypomineralisation

Précis

General dental practitioners should be familiar with the signs of hypomineralised second primary molars (HSPM), as these children are high caries risk and may be more likely to develop molar incisor hypomineralisation (MIH).

Abstract

Background: The term hypomineralised second primary molars (HSPM) describes a prevalent qualitative developmental defect of enamel. Children with HSPM are at a high risk of caries, and are reportedly five times more likely to develop molar incisor hypomineralisation (MIH).

Aetiology: There is an overlap in the development of the second primary molar and the first permanent molar. It is likely that MIH and HSPM have some shared aetiological factors, but in the case of HSPM the insult likely occurred earlier.

Diagnosis: HSPM can be identified as soon as the second primary molar erupts. It has a distinct clinical presentation and many clinical similarities with MIH. HSPM should be differentiated from typical early childhood caries.

Conclusion: Early dental visits for all children would allow early diagnosis of HSPM, which is essential to prevent future problems. Dental teams who work with children should be familiar with the signs of HSPM and use high caries risk preventive strategies, as well as increased vigilance during eruption of the first permanent molars.

Journal of the Irish Dental Association December 2019/January 2020; 65 (6): 340-345

Introduction

Molar incisor hypomineralisation (MIH) is a well-recognised qualitative dental defect often associated with increased treatment needs (**Table 1**).[1] It has an estimated global prevalence of 14.2%,[2] yet the aetiology of MIH is still not fully understood.[1,3] As a result, dentists are not able to predict which children are at an increased risk of developing MIH prior to eruption of the first permanent molars. However, a recent systematic review suggests that detection of a similar hypomineralisation defect in the primary dentition could identify those at an increased risk of developing MIH in the future.[4] This finding is significant, as it may be the only dental risk indicator potentially available to dentists in predicting MIH, and it is something that could be easily identified by routine examination alone (**Figure 1**).

HSPM and MIH

Although by definition MIH refers to hypomineralisation of first permanent molars (FPMs) and incisors,[5,6] MIH-like defects can also affect the tips of canines, second permanent molars and second primary molars.[7] When hypomineralisation occurs in the primary dentition, the second molars are most commonly affected.[8] MIH-like defects affecting second primary molars have been termed "hypomineralised second primary molars" (HSPM).[8] This is defined as "idiopathic hypomineralisation of one to four second primary molars".[8] A recent systematic review determined that the prevalence of HSPM varies from 5-20%, with a mean of approximately 11%.[4] This figure may be underreported because severe caries of the second primary molars could mask the detection of HSPM. Elfrink (2006) reported that occlusal caries is

Dr Rona Leith BA BDentSc DChDent MFD FFD(RCSI)
Assistant Professor in Paediatric Dentistry
Dept. of Public & Child Dental Health
Dublin Dental University Hospital
Lincoln Place, Dublin

Corresponding author: Dr Rona Leith. T: 01-612 7303 E: rona.leith@dental.tcd.ie

TABLE 1: Glossary of key terms.

Molar incisor hypomineralisation (MIH):	Hypomineralisation of systemic origin of one to four first permanent molars frequently associated with affected incisors.[5]
Hypomineralised second primary molars (HSPM):	Idiopathic hypomineralisation of one to four second primary molars.[8,10]
Enamel hypoplasia:	A quantitative defect resulting from disturbance to the ameloblasts during enamel matrix secretion.[5,10]
Enamel hypomineralisation:	A qualitative defect resulting from disturbance during calcification or maturation.[10]

FIGURE 1: HSPM with PEB affecting 65 and MIH with PEB affecting 26. Early identification of HSPM defect in 65 may have predicated the future occurrence of MIH.

significantly more prevalent on second primary molars than first primary molars, and this finding may be attributed to an underlying enamel defect of that particular tooth surface.[8,9] While HSPM is reportedly a prevalent condition, there are very few publications on this topic compared to MIH.[8]

Many studies have reported an association between HSPM and MIH.[4,10-13] One cross-sectional study examining almost 2,000 children reported that MIH was six times more likely to occur in children with HSPM.[13] A recent systematic review, which included five other studies, found that, overall, children with HSPM were approximately five times more likely to develop MIH, and the more molars affected by HSPM, the greater the risk.[4] Interestingly, there may be higher odds of developing MIH for mild HSPM presentations rather than severe.[11] The explanation given is that the disturbance causing milder HSPM is more likely to occur later in their development, which coincides with the earlier, more active mineralisation phases of the FPMs.[10,11] While the absence of HSPM does not exclude the possibility of future MIH,[12] identification of HSPM prior to eruption of the FPMs can be considered a risk indicator. Consequently, children with signs of HSPM should be monitored closely during eruption of their FPMs.[11] Targeting patients who are most likely to develop MIH with early intervention could potentially minimise the complications of MIH[4] or prevent excessive dental visits. It is well documented that children with MIH undergo much more dental treatment and may develop more dental fear as a consequence.[14]

Aetiology of HSPM

Both MIH and HSPM occur as a result of a developmental disturbance of enamel formation during maturation. A common cause could simultaneously affect the FPMs and the second primary molars as they develop in parallel and in a similar jaw location.[10] There is an 11-month overlap in the mineralisation period of the second primary molar and the first permanent molar. The second primary molar begins to calcify at around four months *in utero* and the crown is complete at around 10-11 months of age.[15] The first sign of calcification of the FPM is at birth. By age one the occlusal half of the FPM crown is formed, but it takes much longer for crown mineralisation to complete.[15] Some authors have suggested that the most critical period for enamel defects of FPM is within the first year of life, or more specifically during the first six to seven months, as this coincides with early maturation.[16] Any insult during this timeframe could also affect the second primary molar.

It is likely that MIH and HSPM have some shared aetiological factors, but in the case of HSPM the insult likely occurred earlier in life[10] – perhaps prenatal or perinatal.[3,8,17,18] The aetiology of both MIH and HSPM, while still not fully understood, is likely to be multifactorial and associated with environmental factors and a possible genetic predisposition.[3,18] A recent systematic review concluded that early childhood illness (especially fever) appears to be associated with MIH, and that prenatal or perinatal factors are less frequently involved.[3] The aetiology of HSPM on the other hand may be related to a number of prenatal and perinatal factors, including: maternal alcohol intake; low birth weight; general perinatal morbidity; and, fevers within the first year of life.[18] Others have suggested that perinatal and neonatal factors may be more commonly associated with HSPM rather than prenatal factors.[3] It is important to note that there is significantly less evidence relating to the aetiology of HSPM compared to MIH, and it is clear that more high-quality prospective studies are needed.[3]

Diagnosis of HSPM

Elfrink *et al.* (2015) suggested that the ideal timing for diagnosing HSPM is at five years of age, when the second primary molars have been present in the mouth for approximately two years and the child is more likely to be co-operative.[19] However, signs of HSPM can be detected on second molars as soon as they erupt, so the child should be examined as early as possible. The first step in risk assessment for HSPM is to review the medical history in detail, including the prenatal, perinatal and postnatal period.

Clinically, the European Academy of Paediatric Dentistry (EAPD) diagnostic criteria[6,8] used to diagnose MIH can be adapted for scoring HSPM. The second primary molars should be cleaned and inspected for any of the following signs:

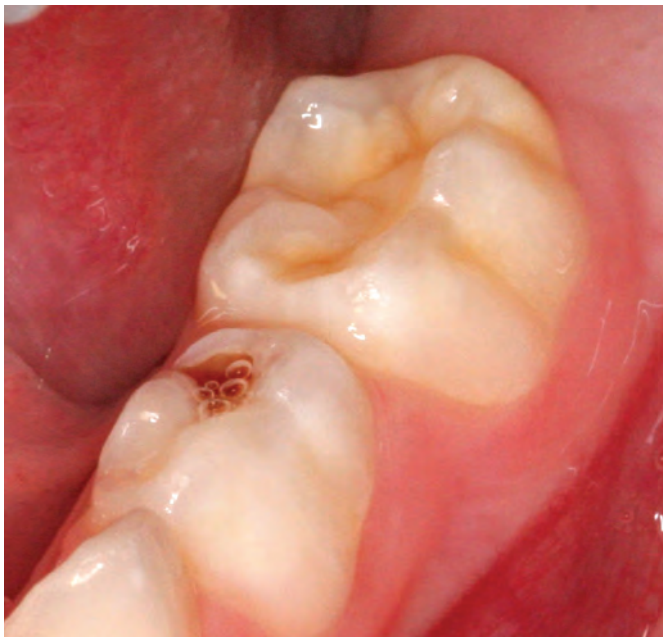

FIGURE 2: Demarcated enamel opacities tooth 75.

FIGURE 3: Atypical caries and PEB tooth 65.

demarcated enamel opacities (**Figure 2**); post-eruptive enamel breakdown (PEB) (**Figure 3**); atypical caries (**Figure 4a**); and, atypical restorations or extractions that do not match the caries pattern of the child.[8,9,20] A demarcated opacity is an area of altered enamel translucency with a clear border, which can be white, yellow or brown in colour.[8] Darker coloured defects indicate more severely hypomineralised enamel with reduced mineral density.[21] Most often, a diagnosis of HSPM is made by detecting demarcated enamel opacities.[11] The defects in HSPM are predominantly located on the occlusal and buccal surfaces, and generally have an asymmetrical distribution (**Figures 4a** and **5a**). Like MIH, the severity and colour can vary within the same mouth.[5,7,10] Once PEB occurs, HSPM can be considered severe.[10] It can also be hard to distinguish hypomineralisation from enamel hypoplasia once the enamel has broken down.[10] Atypical restorations are uncommonly seen as most of the decay in this age group remains untreated.[8,20]

Hypomineralisation is a key risk factor for caries in the primary dentition[10] and children with HSPM are therefore at an increased risk of developing caries[8] (**Figures 4a** and **4b**). Caries prevalence increases in line with HSPM severity and the rate of progression is also accelerated.[20] Furthermore, the chance of presenting with severe untreated caries is greater in children with HSPM than those without.[22] Anecdotal evidence suggests that HSPM is often overlooked as typical early childhood caries (ECC), so the clinician needs to examine the entire primary dentition carefully to differentiate HSPM from the typical pattern of ECC. Once PEB occurs this can be difficult.[20] ECC is generally a symmetrical disease in which teeth that have been exposed to the cariogenic environment the longest tend to be more severely affected. The second primary molar is the last primary tooth to erupt; therefore, breakdown of second primary molars in an otherwise sound dentition should arouse suspicion of a diagnosis of HSPM (**Figures 4a** and **4b**). Likewise, breakdown of the distal surface of a second primary molar before the FPM has erupted (i.e., in the absence of a contact point) is atypical for caries and more likely to suggest HSPM (**Figures 4a** and **4b**). The use of bitewing radiographs to identify interproximal carious lesions is very useful in distinguishing a diagnosis. A vertical bitewing technique may be easier for the child in the primary dentition stage (**Figures 4b** and **5b**).

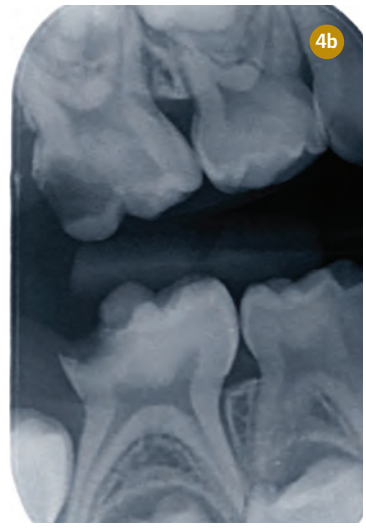

FIGURE 4a: PEB and severe caries affecting 75 and 85, in an otherwise sound dentition.

FIGURE 4b: Right vertical bitewing of patient in Figure 4a. Note first primary molars appear caries free.

FIGURE 5c: Stainless steel crown placed using Hall technique on 85. FS placed on occlusal of 75 using GIC (Fuji triage) to prevent PEB.

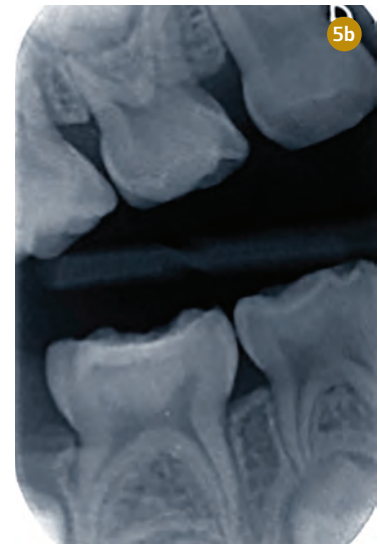

FIGURE 5a: HSPM affecting 75 and 85 with PEB affecting 85. Note the asymmetrical distribution of HSPM. Tooth 85 has been temporised occlusally with a well-placed tooth-coloured RMGIC. Note 74 and 84 are intact.

FIGURE 5b: Right vertical bitewing of patient in Figure 5a following caries control of 85 with RMGIC, taken prior to SSC placement. Note there are no radiographic signs of pulpal pathology.

Management of HSPM

The principles of managing HSPM are similar to those of managing MIH. William *et al*. (2006) suggested a useful six-step management plan for MIH,[23] which can be extrapolated as an overall management strategy for HSPM (**Table 2**).

Preventive

Children with HSPM should be managed using best practice guidelines for those at high caries risk.[24,25] Regular visits give the opportunity to educate parents about both HSPM and the potential implications for the FPMs. Usual high caries risk preventive advice should be delivered at each visit, including dietary counselling and oral hygiene instruction. Parents must assist in brushing their child's teeth twice daily with toothpaste containing 1,000-1,450ppm fluoride.[25] Patients should receive biannual fluoride varnish (22,600ppm) application[24,25] to high-risk sites. Fissure sealants using either resin, glass ionomer cement (GIC) (**Figure 5c**) or resin-modified glass ionomer cement (RMGIC) should be placed before enamel breakdown occurs.[7] Co-operation may be limited in younger patients, so prevention of enamel breakdown is paramount to minimise the need for invasive treatment requiring local anaesthesia.

Operative

If enamel breakdown has occurred, vital primary molars can be stabilised with atraumatic restorative techniques such as a well-placed RMGIC (**Figure 5a**). This type of interim therapeutic restoration is very useful for caries control in children, especially young children.[26] Silver diamine fluoride (SDF) could also be used to arrest caries, but only if there is cavitation and exposed dentine, with no signs of pulpal pathology. SDF has been shown to be an effective and inexpensive method of arresting caries in primary teeth; however, permanent dark discolouration of the carious dentine results.[27] Contemporary guidelines for placement of interim therapeutic restorations and for SDF application are available from the American Academy of Pediatric Dentistry.[26,27]

Composite resin could be used to restore small defects limited to one or two surfaces in HSPM based on their successful use in MIH molars.[7] Difficulties in bonding to hypomineralised enamel in HSPM is likely to be comparable to MIH,[7] so placement under ideal conditions using rubber dam is recommended. However, the use of conventional intracoronal restorations is not always successful due to the atypical location of the defects in HSPM, so full coverage with stainless steel crowns (SSCs) may be more predictable. As conventional SSC preparation using local anaesthesia requires significant co-operation from a young child, the Hall technique[28] may be a useful alternative. In this technique, SSCs are placed without caries removal or local anaesthetic so it is

TABLE 2: Management plan for HSPM (adapted from William et al.).[23]

1. **Risk identification**	Review medical history including prenatal, perinatal, postnatal and infancy period up to age one
2. **Early diagnosis**	Examine teeth as soon as they erupt
3. **Remineralisation and densensitisation**	Dietary counselling; assisted brushing with fluoride toothpaste (1,000-1,450ppm); professionally applied fluoride varnish (22,600ppm F)
4. **Prevention of caries and PEB**	Fissure sealants (GIC, RMGIC or resin); SDF
5. **Restorations or extractions**	RMGIC; composite resin; Hall SSCs; conventional SSCs; extraction
6. **Maintenance**	Regular review (vigilance during eruption of first permanent molars)

much easier for the child to tolerate.[28] Using the Hall technique, SSCs can be placed on second primary molars even before eruption of FPMs[28] (**Figure 5c**). This is a valuable and relatively simple treatment option, assuming any signs of irreversible pulpitis or pulp necrosis have been excluded both clinically and radiographically (**Figure 5b**). A user's manual describing this technique is available.[28]

Once the second primary molars become severely affected or symptomatic, extraction may be the only feasible option. Very often this procedure will necessitate a general anaesthetic due to the young age of the child. While early loss of any primary tooth is undesirable, loss of the second primary molar is particularly unfavourable for the developing occlusion.[29] The subsequent mesial tipping and migration of the FPM[29] results in space loss for the developing second premolar. The earlier the second primary molar is lost, the greater the opportunity for unfavourable drifting of the FPM[29] and future orthodontic disruption.

Maintenance

Children with HSPM are more likely to have caries and poor oral hygiene compared to those without,[30] so regular review to reiterate preventive advice is key. Recall intervals should not exceed four months for these high-risk children, as hypomineralised enamel can deteriorate rapidly.

Conclusion

HSPM is an important diagnosis, which is often overlooked as typical ECC. Dental teams who work with children should be familiar with the signs of HSPM because these children are at high risk of caries, and are more likely to develop MIH. Once identified, high caries risk preventive strategies are essential and practitioners should increase vigilance during eruption of the FPMs. The high reported prevalence of HSPM supports the case for earlier dental visits for all children, to minimise development of future problems and to maximise its potential predictive capacity for MIH.

There is a great need for further prospective studies comparing the prevalence of HSPM and MIH to expand our knowledge of their association.[19] Future research should utilise a standardised protocol as described by Elfrink, to enable comparability of studies from around the world.[19]

References

1. **Schwendicke, F., Elhennawy, K., Reda, S., Bekes, K., Manton, D.J., Krois, J.** Global burden of molar incisor hypomineralization. *J Dent* 2018; 68: 10-18.
2. **Zhao, D., Dong, B., Yu, D., Ren, Q., Sun, Y.** The prevalence of molar incisor hypomineralization: evidence from 70 studies. *Int J Paediatr Dent* 2018; 28 (2): 170-179.
3. **Silva, M.J., Scurrah, K.J., Craig, J.M., Manton, D.J., Kilpatrick, N.** Etiology of molar incisor hypomineralization. A systematic review. *Community Dent Oral Epidemiol* 2016; 44 (4): 342-353.
4. **Garot, E., Denis, A., Delbos, Y., Manton, D., Silva, M., Rouas, P.** Are hypomineralised lesions on second primary molars (HSPM) a predictive sign of molar incisor hypomineralisation (MIH)? A systematic review and meta-analysis. *J Dent* 2018; 72: 8-13.
5. **Weerheijm, K.L., Jälevik, B., Alaluusua, S.** Molar incisor hypomineralisation. *Caries Res* 2001; 35 (5): 390-391.
6. **Weerheijm, K.L., Duggal, M., Mejäre, I., Papagiannoulis, L., Koch, G., Martens, L.C., et al.** Judgement criteria for molar incisor hypomineralisation (MIH) in epidemiologic studies: a summary of the European meeting on MIH held in Athens, 2003. *Eur J Paediatr Dent* 2003; 4 (3): 110-113.
7. **Lygidakis, N.A., Wong, F., Jälevik, B., Vierrou, A.M., Alaluusua, S., Espelid, I.** Best Clinical Practice Guidance for clinicians dealing with children presenting with Molar-Incisor-Hypomineralisation (MIH): An EAPD Policy Document. *Eur Arch Paediatr Dent* 2010; 11 (2): 75-81.
8. **Elfrink, M.E., Schuller, A.A., Weerheijm, K.L., Veerkamp, J.S.** Hypomineralized second primary molars: prevalence data in Dutch 5-year-olds. *Caries Res* 2008; 42 (4): 282-285.
9. **Elfrink, M.E., Veerkamp, J.S., Kalsbeek, H.** Caries pattern in primary molars in Dutch 5-year-old children. *Eur Arch Paediatr Dent* 2006; 7 (4): 236-240.
10. **Elfrink, M.E., ten Cate, J.M., Jaddoe, V.W., Hofman, A., Moll, H.A., Veerkamp, J.S.** Deciduous molar hypomineralization and molar incisor hypomineralization. *J Dent Res* 2012; 91 (6): 551-555.
11. **Mittal, N., Sharma, B.B.** Hypomineralised second primary molars: prevalence, defect characteristics and possible association with molar incisor hypomineralisation in Indian children. *Eur Arch Paediatr Dent* 2015; 16 (6): 441-447.
12. **Negre-Barber, A., Montiel-Company, J.M., Boronat-Catalá, M., Catalá-Pizarro, M., Almerich-Silla, J.M.** Hypomineralized second primary molars as predictor of molar incisor hypomineralization. *Sci Rep* 2016; 6: 31929.
13. **da Silva Figueiredo Sé, M.J., Ribeiro, A.P.D., Dos Santos-Pinto, L.A.M., de Cassia Loiola Cordeiro, R., Cabral, R.N., Leal, S.C.** Are hypomineralized primary molars and canines associated with molar-incisor hypomineralization? *Paediatr Dent* 2017; 39 (7): 445-449.
14. **Jälevik, B., Klingberg, G.A.** 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 (1): 24-32.
15. **Shour, I., Massler, M.** The development of the human dentition. *JADA* 1941; 28: 1153-1160.
16. **Fagrell, T.G., Salmon, P., Melin, L., Norén, J.G.** Onset of molar incisor hypomineralization (MIH). *Swed Dent J* 2013; 37 (2): 61-70.
17. **Aine, L., Backström, M.C., Mäki, R., Kuusela, A.L., Koivisto, A.M., Ikonen, R.S., et al.** Enamel defects in primary and permanent teeth of children born prematurely. *J Oral Pathol Med* 2000; 29 (8): 403-409.
18. **Elfrink, M.E., Moll, H.A., Kiefte-de Jong, J.C., Jaddoe, V.W., Hofman, A., ten Cate, J.M., et al**. Pre- and postnatal determinants of deciduous molar hypomineralisation in 6-year-old children. The generation R study. *PLoS One* 2014; 9 (7): e91057.
19. **Elfrink, M.E., Ghanim, A., Manton, D.J., Weerheijm, K.L.** Standardised studies on molar incisor hypomineralisation (MIH) and hypomineralised second primary molars (HSPM): a need. *Eur Arch Paediatr Dent* 2015; 16 (3): 247-255.
20. **Ghanim, A., Manton, D., Mariño, R., Morgan, M., Bailey, D.** Prevalence of demarcated hypomineralisation defects in second primary molars in Iraqi children. *Int J Paediatr Dent* 2013; 23 (1): 48-55.
21. **Elfrink, M.E., ten Cate, J.M., van Ruijven, L.J., Veerkamp, J.S.** Mineral content in teeth with deciduous molar hypomineralisation (DMH). *J Dent* 2013; 41 (11): 974-978.
22. **Gambetta-Tessini, K., Mariño, R., Ghanim, A., Calache, H., Manton, D.J.** Carious lesion severity and demarcated hypomineralized lesions of tooth enamel in schoolchildren from Melbourne, Australia. *Aust Dent J* 2018 Jun 7. [epub ahead of print]
23. **William, V., Messer, L.B., Burrow, M.F.** Molar incisor-hypomineralisation: review and recommendations for clinical management. *Pediatr Dent* 2006; 28 (3): 224-232.

24. **Scottish Intercollegiate Guidelines Network.** Dental interventions to prevent caries in children. A national clinical guideline. SIGN 2014. Available at: https://www.sign.ac.uk/assets/sign138.pdf.
25. **Health Service Executive, University College Cork, Health Research Board.** Strategies to Prevent Caries in Children and Adolescents. Summary Guideline. Available from: https://www.dentalhealth.ie/assets/files/pdf/shortguidelines.pdf.
26. **American Academy of Pediatric Dentistry.** AAPD Policy on Interim Therapeutic Restorations, 2017. Available from: http://www.aapd.org/media/policies_guidelines/p_itr.pdf.
27. **American Academy of Pediatric Dentistry.** Use of silver diamine fluoride for dental caries management in children and adolescents including those with special health care needs. 2017. Available from: https://www.aapd.org/media/policies_guidelines/g_sdf.pdf.
28. **University of Dundee.** The Hall Technique. A minimal intervention, child centred approach to managing the carious primary molar. Available from: https://dentistry.dundee.ac.uk/files/3M_93C%20HallTechGuide2191110.pdf.
29. **Royal College of Surgeons.** Extraction of primary teeth – balance and compensation. RCS Eng. Available from: https://www.rcseng.ac.uk/-/media/files/rcs/fds/publications/extractp.pdf.
30. **Oyedele, T.A., Folayan, M.O., Oziegbe, E.O.** Hypomineralised second primary molars: prevalence, pattern and associated co morbidities in 8- to 10-year-old children in Ile-Ife, Nigeria. *BMC Oral Health* 2016; 16 (1): 65.

CPD questions

To claim CPD points, go to the MEMBERS' SECTION of www.dentist.ie and answer the following questions:

1. Which of the following best describes the enamel defect in HSPM?

- ○ A: A quantitative defect with demarcated enamel opacities
- ○ B: A quantitative defect with diffuse enamel opacities
- ○ C: A qualitative defect with demarcated enamel opacities
- ○ D: A qualitative defect with diffuse enamel opacities

2. The typical clinical presentation of HSPM is best described by which one of the following?

- ○ A: Chronological hypoplasia along the occlusal and buccal surfaces of the second primary molar
- ○ B: Symmetrical demarcated opacities along the cervical margin of the second primary molar
- ○ C: Asymmetrical demarcated enamel opacities on the occlusal and buccal surfaces of the second primary molar
- ○ D: Demineralisation along the gingival margin of the first and second primary molar

3. Which of the following is incorrect regarding HSPM?

- ○ A: The absence of HSPM excludes the possibility of future MIH
- ○ B: Identification of HSPM prior to eruption of the first permanent molars can be considered a risk factor for MIH
- ○ C: Hypomineralisation is an important risk factor for caries in the primary dentition
- ○ D: Mild HSPM may be at higher odds to develop MIH than severe presentations

IDA irish dental association CPD