THE PREVALENCE AND SEVERITY OF MOLAR INCISOR HYPOMINERALISATION IN DUBAI, UAE, A CROSS SECTIONAL STUDY

By

Dr. Ghada Hussain

BDS, Trinity College Dublin, 2004

BA, Trinity College Dublin, 2004

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Mohammed Bin Rashid University of Medicine and Health Sciences
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ABSTRACT

THE PREVALENCE OF MOLAR INCISOR HYPOMINERALISATION IN DUBAI CITY IN UAE, A CROSS SECTIONAL STUDY

Dr. Ghada Hussain, BDS, BA

Supervisors: Associate Professor Manal Al Halabi and Associate Professor Mawlood Kowash

Aim: The aim of this study was to evaluate the prevalence and severity of Molar Incisor Hypominerlisation (MIH) among school children in Dubai, United Arab Emirates (UAE).

Materials and Methods:

A randomised cluster sample of 8 to 12 year-old children had their first permanent molars and permanent incisors (index teeth) evaluated for prevalence and severity of MIH using the criteria of the European Academy of Paediatric Dentistry. The examinations were conducted at public schools by two calibrated examiners. A total of 342 children (mean age = 9.46) were randomly selected from public schools in Dubai and Hatta (rural area).

Results:

The prevalence of MIH was 27.2 % in Dubai, (93 out of 342 children); there were no significant differences between Dubai and Hatta. The prevalence of MIH was significantly greater in girls (32.6%) compared to (18.1%) in boys with a p-value of 0.002. The prevalence of MH was higher than MIH: 61 out of 93 children (65.6%) had MH, compared to 32 out of 93 children (34.4%)

who had MIH. MH in maxillary molars was significantly higher than mandibular molars, 20.8% compared to 14.6% (p= <0.005).

Maxillary incisors were effected more by MIH 8.8% compare to mandibular incisors (0.9%) (p= <0.001).

The presence of demarcated opacities was significantly higher in girls than boys (p = 0.002). Moreover, a large majority of the children with MIH in the present study (53%) presented with mild defects, (17%) moderate defect and (30%) severe defects.

Conclusions:

The prevalence of MIH in school children in Dubai was 27.2 percent. Location and age appeared to have no significant correlation with MIH except in gender. Girls had more MIH than boys (P=0.002), which warrants further research.

DEDICATION

This thesis is dedicated to my parents

For their endless love, support and encouragement

DECLARATION

I declare that all the content of the thesis is my own work. There is no conflict of interest with any other entity or organization.

Name: Ghada Hussain

Signature:

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List of Abbreviations

MIH = Molar-Incisor-Hypomineralisation

MH = Molar Hypomineralisation

FPM = First Permanent Molar

PEB =Post Eruptive enamel Breakdown

AR= Atypical Restoration

DDE= Developmental Defects of Enamel

UR6 = Upper Right first molar

UR2 = Upper Right lateral incisor

UR1 = Upper Right central incisor

UL1 = Upper Left central incisor

UL2 = Upper Left lateral incisor

UL6 = Upper Left first molar

LL6 = Lower Left first molar

LL2 = Lower Left lateral incisor

LL1 = lower Left central incisor

LR1 = Lower Right central incisor

LR2 =Lower Right Lateral incisor

LR6 =Lower Right first molar

UAE = United Arab Emirates

1. Introduction

Molar-Incisor-Hypomineralisation (MIH) may otherwise be defined as the "developmental enamel hypomineralisation of unknown aetiology of the first permanent molars (FPM) with frequent involvement of permanent incisors" [1]. Regardless of the fact that MIH is thought to be a dental/clinical problem worldwide [2], the data available regarding the existence and severity of MIH remains inadequate. According to the data that has been published, the prevalence of MIH is context-dependant and differs between counties and years of birth; the variation is between 2.4% to 40.2%[3]. Some of these reports have included several birth years grouped together (Leppäniemi et al., 2001)[4] or only one birth cohort [(Jälevik et al., 2001)[5]; (Weerheijm et al., 2001b)[1]; (Calderara et al., 2005)[6]; (Fleita et al., 2006)[7]]. From these studies, it can be determined that MIH is multi-factorial in nature and as such no one clear cause for MIH can be identified. Due to the fact that epidemiological surveys regarding caries prevalence conducted on the national-level do not include screening for MIH, studies which compare the prevalence of MIH in several birth cohorts are lacking. The most prominent and relevant studies of MIH were carried out in European countries with the prevalence of MIH ranging between 3.6% and 19.3 %[3,4,5,8,9,10,11,12]. Other studies have documented a 22% prevalence of MIH in Australia[13], a 14.8% prevalence in Turkey[14], 2.9% in Libya[15] and 13.70% in Kenya[16].

The aim of the present study was to evaluate the prevalence of MIH in a population of children in Dubai, United Arab Emirates (UAE). It also sought to highlight the clinical characteristics and the severity of the affected teeth. In an attempt to determine the overall clinical findings of the disorder, correlations between the number, type and severity of affected teeth was also recorded and discussed.

2. Literature Review

2.1 Definition

A term first suggested by Weeheijm (2001), Molar Incisor Hypomineralisation (MIH) is defined as the "hypomineralisation of systemic origin of one to four permanent molars frequently associated with affected incisors"[1]. Other authors[10,17,18] also commonly refer to MIH as 'cheese molars', 'enamel opacities', 'non-fluoride hypominerlasation',[19] and 'idiopathic enamel hypomineralisation'. On the other hand, Molar Hypomineralisation (MH) describes "hypomineralsation of one to four first permanent molars with no affected incisors"[20]. Both MIH and MH form a part of a hypomineralisation spectrum, where MIH is the more severe form[20].

2.2 Prevalence

Most studies regarding the prevalence of MIH were conducted in European countries and demonstrated the prevalence to be between 3.6% and 19.3 %[3,5,1,8,18,17,10,21,22]. Other studies reported a prevalence rate of 22% in Australia[13], a rate of 14.8% in Turkey [3], 2.9 % in Libya[7] 13.70% in Kenya[16].

A study conducted in the South American country of Brazil revealed a very high prevalence of MIH, scoring at a 40.2 %[23]. Comparatively, a study conducted in East Asia, and Hong Kong in particular, reported a prevalence of 2.8%[3]. There is a lack of published data in North America and the Middle East. There are a few studies, however, in Iraq and in Jordan which report the prevalence of MIH to be 18.6 %[24] and 17.6 %[25] respectively.

Due to the variation in number of factors including study methodologies, population base samples, age, calibration, severity, wet/dry examination criteria, the tooth number and their type, the examination method and the size of the defect[3], it is very difficult to compare the prevalence of MIH in differing contexts.

The types of studies which are conducted to show the prevalence of MIH were mainly cross-sectional, retrospective or prospective, with disease progression from age 8 till 18 years old.

Some studies used the modified Developmental Defect of Enamel tooth index (mDD) criteria presented by FDI in 1992 [26]. According to this criterion, Developmental Defects of Enamel (DDE) are classified as demarcated or diffused opacities and hypoplasia. Enamel opacity is defined as a qualitative defect in enamel and an abnormality in the translucency of enamel. Enamel Hypoplasia is defined as a quantitative defect with reduced thickness in enamel. The mDD index is found to be time-consuming and more difficult to analyze[26]. Koch et al (1987)[8] published a study describing enamel defects in terms of color and surface changes. Alaluusua and coworkers (1996 a, b) published two studies that looked at enamel defects in First Permanent Molars (FPM) which excluded fluorosis, hypoplasia and defects related to major disturbances in general health. The degree of the severity and size of the defects differentiated and registered as: firstly, severe (loss of enamel with need for restoration, atypical restoration), secondly, moderate (loss of enamel), and lastly, mild (color change).The sizes were recorded as: large (≥4.5 mm), moderate (≈3.5mm), small (≈2mm) [27].

The EAPD seminar on MIH held in Athens in 2003, agreed on the following criteria for the evaluation of MIH[2]:

- Permanent first molars and incisors (12 index teeth) should be examined
- Examination for MIH should be performed on wet teeth after cleaning

- Eight years of age is the best time for examination

Furthermore, each tooth should be examined for: Absence or presence of demarcated opacities, post eruptive enamel breakdown (PEB), atypical restoration (AR), extraction due to MIH, as well as the failure of eruption of a molar or incisor.

Most MIH-relevant studies report the frequency of MIH within a specific group rather than the mere prevalence of MIH. The prevalence of MIH pertaining to different age groups within three different studies (Koch et al., 1987[8]; Dietrich et al., 2003[28]; Kukleva et al., 2008[29]) showed considerable variation between the groups.

Some examples of epidemiological studies for MIH, in Table 1 adapted from B. Jälevik,2010 [3]:

Table 1

Study	Country	Age	Sample Size	Criteria	Calibra ti-on	Wet/Dr y	Dropouts	Size	Seve rity	Frequency
Alaluusua et al. 1996a[30]	Finland	6-7yrs	102	Dental defects, major disturbance to general health and fluorosis excluded.	NR	NR	NR	≥ 2mm	3	17%
Balmer et al. 2005[31]	UK	8-16yr	25	mDDE	Yes	Wet	NR	NR	NR	40%
	Australia		25							44%
Arrow 2008[32]	Australia	7yr	511	mDDE	Yes	Dry	R	NR	NR	22%
Cho et al 2008[33]	Hong Kong	11-14yr	2,635	EAPD 2003	Yes	Wet	NR	NR	NR	2.8%
Fleita et al 2006[15]	Libya	7-9yr	378	mDDE	Yes	Wet	NR	≥ 2mm	3	2.9%
Kukleva et al. 2008[29]	Bulgaria	7-14yr	2970, 370 in each age group	EAPD 2003	NR	Wet	NR	NR	NR	2.4%-7.85, mean 3.6%
Lygidakis et al. 2008[34]	Greece	5.5-12yr	3518	EAPD 2003	Yes	NR	NR	Clearly visible	2	10.2%
Zagdwon et al, 2002[35]	UK	7yr	307	mDDE	Yes	Wet	R	NR	NR	14.6%
Ghanim et al,2011[36]	Iraq	7-9yrs	823	EAPD 2003	R	Dry	R	≥ 2mm	3	21.5%
Zawaideh et al,2011[25]	Jordan	7-9 yrs	3666	EAPD 2003	NR	Dry	R	NR	3	17.6%

Ghanim et al, 2014[37]	Iran	9-11 yrs	810	EAPD 2003	R	Wet	NR	NR	NR	20.2%
Allazzam et al ,2014[38]	KSA(Jedda)	8-12yrs	265	EAPD 2003	R	NR	NR	NR	NR	8.6%
Kevrekidou et al 2015	Greece	8 &14yrs	1197 1156	EAPD 2003	R	Wet	NR	NR	3	21%
De Lima et al 2015[39]	Brazil	11 -14 yrs	594	EAPD 2003	R	Wet	R	≥ 1mm	3	18.4%
Yannam et al 2016[40]	India	8-12y	2864	EAPD 2003	NR	NR	NR	≥2mm	NR	9.7%
Subramaniam et al 2016[41]	India	7-9 yrs	2500	EAPD 2003	R	Wet	NR	NR	NR	0.48%

R=Reported, NR= Not Reported

It is indeed necessary to have a well-defined study group which is an accurate representation of a given population. The rate of dropout must be described in addition to the recruitment procedures[3]. Each age should preferably be reported separately with at least 100 children in each group[3]. The EAPD recommends the best age to carry out the examination to be 8 years of age. The size of the defect should be recorded and it has been proposed not to record any defects smaller than 1 mm[26]. This is in order to eliminate the increased risks of misdiagnosis of e.g. white spot lesions, white cuspal ridges or a patchy appearance of *amelogenesis imperfecta* or fluorosis. The criteria of examination, whether wet or dry, should also be reported. Training and calibration is also very important. The degree of disturbance appears to vary with the number of affected teeth [1,42] but not with the prevalence of MIH[43]. It is for that reason that it is also important to report the severity of the defects in a consistent manner. It would be beneficial to report the number of index teeth affected in each child with MIH, as the eruption stages will influence this result[34,43,44].

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2.3 Pathogenesis

Tooth enamel is resultant from the ectoderm and is made by ameloblasts which are differentiate from the inner enamel epithelial cells of the dental organ. Enamel formation can be divided into three stages: matrix formation, initial calcification and final maturation[45]. In the first stage of matrix formation, and in particular during the 20th to the 32nd week in ueteo, the enamel of the permanent first molar and the permanent incisors begins[46]. Once the first layer of dentin is formed, the ameloblasts start their secretory function. The now partially mineralized enamel matrix includes a large amount of matrix proteins within which long thin ribbons of enamel mineral is laid, mainly hydroxyapatite. The secretory phase starts at cuspal tips and spreads down the cuspal slops. There are a number of things that occur during the secretory phase. Throughout the phase, the enamel crystals grow in length whereas the enamel layers grow in thickness. The mineral content of secretory enamel is approximately 10–20% by volume, with the remaining portion occupied by matrix protein and water. Whence the full thickness of enamel has been deposited, the secretory ameloblasts are transformed into maturation stage ameloblasts. This occurs in the second stage of initial calcification. The newly formed maturation stage ameloblasts are responsible in the processes of matrix degradation and the mineralisation of the enamel. Although the "maturation phase" starts at birth, the processes of mineralisation continue during the first year after birth[45]. During the last stage, the final mineralisation of the enamel occurs whilst regulated by the mature ameloblasts. Alongside the growth in the thickness and width of the crystallites, the enamel layer is hardened. This results in mineralised tissue whose weight is consisting of more than 95% mineral. The entire process of enamel formation occurs over the span of one thousand days[47], where two thirds of this time is devoted to the maturation stage of amelogenesis.

The maturation of the enamel involves: firstly, "the removal of acid-labile mineral, secondly, replacement with more acid-resistant apatite, [thirdly] an influx of calcium and phosphate ions, increasing the crystal width and thickness"[48]. The disturbed resorptive potential of ameloblasts and the inhibition of proteolytic enzymes often lead to hypomineralisation. This process sometimes leads to the retention of protein (particularly amelogenin) and furthermore an interference both the process of the maturation of enamel as well as the growth of crystals [49,45,50,51]. In order for there to exist a normal deposition of apatite alongside a normal growth of crystallites, the regulation of pH during mineralization is considered crucial[52]. Investigating the relationship between enamel matrix pH during the maturation phase in cystic fibrosis mice, Sui et al.[52] has reported that, when investigating the relationship between enamel matrix pH during the maturation phase in cystic fibrosis mice, the growth of crystals was disrupted by a reduced enamel matrix pH. Because of this, protein retention and hypomineralisation were a result. The above study also recorded that after respiratory acidosis, hypomineralisation followed suit[53]. Theoretically, such conditions affecting matrix pH during enamel maturation may predispose a patient to develop MIH[52]. Furthermore, hypominerlised enamel may also be caused by the lack of calcium phosphate[54]. Using secondary ion mass spectrometry and X-ray microanalysis, the increased severity of hypomineralisation displayed a positive correlation with an increase in carbon concentration and a contrary decrease in the concentration of calcium and phosphorus. The result of this was enamel which contained significantly lowered calcium/phosphorous ratios[55]. Thus, impaired calcium metabolism may have a role in the development of hypomineralised enamel.

Hardness, porosity, and mineral content may be the reflection of hypomineralised enamel defects. As opposed to white defects and normal enamel, yellow-brown defects display lower Knoop hardness values and greater porosity[9,56]. Unaffected enamel also, through nano-

indentation studies, show substantially less values for hardness and modulus of elasticity[57]. Under scanning electron microscopy, the defects showed increased porosity and disorganized rod structure of fractured surfaces[57].

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 hypermineralised with post-eruptive maturation[58]. An overall reduction in mineral concentration (of about 5%) as well as lower calcium and phosphorus ratios are shown in sound enamel of hypomineralised molars, indicating that the entire crown is affected to some extent[58,55].

Although the development of teeth is mainly controlled by genetics, environmental disturbances definitely play a role. During the secretory stage, systemic factors may disturb the ameloblasts, thereby causing a restriction of crystal elongation and resulting in pathologically thin or hypoplastic enamel[59]. During the transitional and maturation stage of amelogenesis, disturbances cause pathologically soft (hypomaturated, hypomineralised) enamel of normal thickness. During the early stages of maturation, ameloblasts are highly sensitive to environmental disturbances[45]. Histological and radiographic studies show that the first signs of mineralisation are seen in the cusp tips around or soon after birth[60,61]. Around the age of six months the four cusps become united[61]. In the end of the first year deposition of the enamel matrix is completed in the occlusal half of the crown and maturation is ongoing (early maturation phase). No morphological changes in the dentine were found by polarized light microscopy except for the presence of interglobular dentine under the affected enamel. Also an overall reduction of about 5% was measured in the mineral concentrations of both the apparently normal enamel and dentine within the MIH teeth compared with those of control teeth studied by an x-ray microtomography[58]. Enamel maturation in the FPM takes several years (later maturation stage), hypomineralisation may develop later.

Rodd et al (2007)[62] found that there was a greater innervation density in the pulp horn and subodontoblast layer in MIH teeth compared to sound enamel teeth. Further, where enamel loss had occurred, immune cells were found abundant in MIH pulps. Also, he found that there was an increased vascularity in hypersensitive MIH teeth[62].

In an in vitro study conducted by Fagrell and co-workers (2008)[63] that studied MH teeth, it was found that the oral bacteria in dentinal tubules can penetrate through and into the hypominerlised enamel into dentin which may contribute to hypersensitivity of MIH teeth[63]. Additionally, the level of carbon and magnesium is higher in the dentin below affected enamel, as opposed to that below sound enamel[64].

2.4 Clinical Diagnosis

The clinical appearance of MIH and the diagnostic criteria are as follows:

Permanent first molars and incisors

At least one of all four first permanent molars (FPM) may show enamel hypomineralisation with or without permanent incisors involvement. One FPM must be affected in order to identify and diagnose MIH. Other teeth may show defects like in second primary molars and tip of the canines. The defects is more severe when more molars and incisors are affected[65].

Demarcated opacities

Clear and demarcated opacities are often seen at the occlusal and buccal part of the crown of the affected teeth. The defects are demarcated opacities differ in both size and colour. The colour of the opacities may be white, creamy or yellow to brownish. It is recommended that defects less than 1 mm are not to be reported[65].

Enamel disintegration[65]

There is some difference in the degree of porosity within the hypomineralised opaque areas. Enamel which is severely affected easily breaks down by masticatory forces, which leads to unprotected dentine and rapid caries development (post-eruptive breakdown).

Atypical restorations

FPM and incisors with restorations showing alike extensions of MIH are recommended to be refereed as affected.

Tooth sensitivity

Affected teeth are often sensitive, ranging from a mild response to external stimuli to spontaneous hypersensitivity. These teeth are usually difficult to anaesthetize[65].

Extracted teeth

It is not possible to diagnose MIH unless the extracted tooth in question was defined as having MIH in the notes or the records, or if there are demarcated opacities on the other FPM.

Recording the severity of the defects

Severity should be recorded as mild or severe. In mild cases there are demarcated enamel opacities without enamel breakdown, infrequent sensitivity to external stimuli e.g. water/air but not during brushing and only slight aesthetic concerns on discolouration of the incisors. In severe cases there are demarcated enamel opacities with breakdown, caries, spontaneous hypersensitivity affecting eating and brushing. In addition to major aesthetic concerns that may have socio-psychological impact on the child.

2.5 Aetiology

There is no conclusive evidence of the causative factors that contribute to enamel hypomineralisation. Many factors were analysed in order to find their correlation to MIH.

Prenatal period

Freden et al (1980)[66] reported that urinary tract infections during the last trimester were associated with lesions similar to MIH[67].

Whatling and Fearne (2008)[67]and Lygidakis et al., (2008)[34] have reported that MIH in children with mothers that have medical problems is more common.

Perinatal period

In a study conducted by Lygidakis et al (2008)[34] MIH was most commonly witnessed in perinatal difficulties such as Caesarean section, twining, prolonged delivery or premature birth.

Whatling and Fearne (2008)[68] and Diedrich et al (2003)[69] reported no linkage between MIH and perinatal problems.

Hypoxia

It has been suggested by many authors that the lack of oxygen available for amelobasts may cause opacities in molars and incisors[70,71,72].

Premature children whose birth weight was less than 1.500g had more opacities but not more hypoplastic molars than control mature children (Seow, 1996)[70]. An animal study conducted by Baumgardner et al (1996)[73] reported that placing rats in a hypobaric chamber at 0.5 atm for 24 hours resulted in little variation in mature ameloblasts. When compared to control

animals, hypoxic disturbance was noticed in the cells of the pulp and surrounding periodontium. However a study by Whitford et al (1995)[53] showed respiratory acidosis (hypoxia) induced 10% CO_2 for 42 days caused enamel hypomineralisation in rat incisors.

Hypocalcaemia

Hypocalacemia can be the result of several various factors/conditions such as maternal diabetes, vitamin D deficiency during the prenatal and/or perinatal period and prematurity. The calcium (Ca) level in MIH teeth is low, suggesting that this could be caused by imparted calcium metabolism in amelobalsts[1]. In children who have nutritional rickets as well as hypocalcaemia, there was more enamel hypoplasia, rather than hypomineralisation, that was recorded[74]. Results from an animal study showed that within several weeks after the start of a calcium-deficient diet, there was a decrease in dentine thickness, and a normal enamel, within the subjects[75]. A period less than 3 weeks with low calcium diet resulted in morphological alterations.

Another study by Bonucci et al (1994)[76] found that rats nursed by low calcium diets developed hypomineralsed enamel in their incisors.

Postnatal period

Tapias Ledesma et al. (2003)[77] showed a strong correlation between frequent medical care for the child and enamel defects in first permanent molars during the first 4 years of life.

Lygidakis et al. (2008)[34] reported that during the first year of infancy, post-natal problems were more common in children with MIH than those without. Another study found that children with MIH had a higher history of medical problems in their first 3 years of life than children without MIH[14].

Childhood Illness/High Fever

Illness such as asthma[1], pneumonia[1,78], otitis media[78] ,chickenpox[67] and urinary tract infections[79] have been positively associated with MIH. However, there does seem to be various contradicting results in other studies[1,67].

Fever is a common symptom of childhood illness. In an experimental study, Turpentine, an exogeneous pyrogen, induced enamel hypomineralisation in rat incisors[80]. The test showed that rat temperature was 1.5°C higher than the control and the fever lasted for 57 hours. Five days into the study, a radiolucent line along with an incremental line were seen in microradiographs, thereby indicating enamel formation was influenced by high fever[59].

Antibiotics

It is not possible to determine whether illness or fever or taking antibiotics causative factors of MIH or whether both are involved[59] . However, an increased risk of MIH has been associated with using amoxicillin during the first year of life[81]. A study by Whatling and Fearne (2008)[67] concluded that children who had amoxicillin during the first four years of age were more commonly displaying MIH, rather those children who had been treated with mixed antibiotics which include amoxicillin. A classic study by Koch et al (1987)[8] showed that the range of MIH in children born in Sweden in 1970 was (15.4%) more than children born in 1966, 1969, 1971, 1972 and 1974 (4.4%-7.3%). Amoxicillin was only available in Sweden post-1975. Therefore, it could not be said that amoxicillin is a cause of MIH[8].

The effect of amoxicillin in tooth development was studied by a tooth culture in mice[81]. They were dissected on 18th day "embryonic", when enamel matrix secretion was about to start. The teeth were cultured, some with and some without amoxicillin (100microg/ml - 4mg/ml) for 10 days, after which they were transferred to the maturation stage. The ameloblasts of

molars exposed to amoxicillin (4mg/ml) remained elongated after 10days culture. This resulted in a different pattern of amelogensis which may interfere with mineralisation.

Children with MIH were found to have used erythromycin in their first year of life[81]. There is a higher risk of forming enamel defects when using macrolides in the first 3 years of life[79].

In an experimental study on rodents conducted by Abe et al. (2003)[82], macrolides were administrated orally at dose of 5,000mg/kg/day for 5 weeks to recreate a high dose as well as a long-lasting exposure. By using histological methods, changes were seen within the ameloblasts at both the transitional and the maturation stage. In addition, the hypomineralisation zone was seen in the incisors after 4 weeks indicating toxicity effect of macrolides.

Environmental toxicants

An exposure to high level of dioxins and polychlorinated biphenyls (PCBs) in early childhood was found to be associated with hypoplasia/ demarcated opacities[83,84]. The higher the dose the higher the prevalence of MIH, Dose-response relationship between pollutant serum concentration and developmental enamel defects[83]. The prevalence of MIH in children living in Slovenia was higher in PCB contaminated areas as opposed to those in control areas[85]. One study found a significant correlation between MIH and the exposure of the children to dioxins via the mother's milk[86]. One study by Laisi et al (2008)[87] found there to be no correlation between a low level of dioxin exposure and the prevalence of MIH. The degradation and/or removal of enamel matrix proteins in developing molars of rat pups was affected by the most toxic dioxin congener, 2, 3, 7, 8-tetrachlorodibenzo-para-dioxin (TCDD) which is transferred to the rat pups via their dams' milk . The removal of enamel

matrix is essential for the completion of enamel mineralisation; this apparently leads to disturbances in mineralisation[88].

A recent experimental study has shown a possible relationship of endocrine-disturbing chemicals such as bisphenol A (BPA) with MIH. This occurs through an effect on the genes regulating proteases, responsible for the protein removal from the enamel matrix[89].

Breast –feeding

A Finnish study by Alaluusua et al (1996a)[27] found that the longer the breastfeeding, the more risk of developing MIH. Various authors have suggested that the toxins in human milk may have been involved; however, as the levels of toxins in milk, such as dioxins, were not measured, the role of pollutants remained theoretical. Results from other European studies have not suggested the contrary and have not found an association between the duration of breast-feeding and the prevalence of MIH[1,67].

Additionally, long breast feeding with late introduction of gruel and formula milk increased the risk to develop severe hypomineralisation by 5 times, indicating an aetiological factor involving nutrition[8].

Fluorides

Fluoride is believed to affect enamel crystal formation mainly during the maturation stage which thereby induces defects described as “diffuse opacities”. The FDI index is most often used in the screening and differentiation of demarcated opacities on one hand, and diffuse opacities on the other hand[90]. With regards to demarcated opacities, there has been no association found between fluoride exposure and demarcated opacities[91,92,93,94]. In three other

studies the association between fluoride supplementation and MIH was studied and there was no significant association found[8,27,67].

Genetics

A recent experimental study has shown that several genes implicated with amelogenesis appear to contribute to the presence of MIH[89]. Previous clinical studies in the population of twins has shown a possible genetic influence on MIH[72,95]. A recent clinical study of twins noticed the possible genetic predisposition in MIH[96].

2.6 Clinical challenges

Loss of tooth substance

Al-Dobiyan et al. (2006)[97] have found that MIH enamel has lower hardness values, less Ca levels, and more carbon levels when compared to normal enamel, hence the post-eruptive breakdown. Bacteria were found deep in the porous MIH close to enamel–dentin junction (EDJ), which increases the risk of caries[63]. As the molars are more susceptible to caries and the accumulation of plaque, MIH increases the need for dental treatment[4]. Children with MIH are ten times more likely to get dental treatment when compared to children without MIH, leading to them being more fearful in the dental environment[12]. Children may be facing great discomfort as, due to the porous surface of enamel alongside exposed dentine and children may be more susceptible to sensitivity to cold air, food, warm water and tooth brushing[12].

Sensitivity

Reported sensitivity may reflect underlying pulpal inflammation (an increase in expression of transient reception potential ion channel) (TRVI)[62]. It has been found that MIH teeth demonstrate a significant increase in neural density in the pulp horn and subodontoblastic region accompanied by more immune cell accumulation especially with post-eruptive enamel loss. In addition, a significant increase in vascularity in sensitive MIH teeth was observed. Importantly, these factors may lead to difficulty in achieving local analgesia[62].

Appearance

A study conducted in England interviewed people aged 10-15 years with Developmental Defects of Enamel (DDE). These interviewees were asked them about the impact of DDE; the researchers found that the variation of the impact of DDE was related to sense-of-self rather than enamel defects[98].

2.7 Treatment Modalities in Children with MIH

William et al (2006a)[19] has proposed and called for a six-step management approach for MIH, Involved in these six steps are: risk identification, early diagnosis, remineralisation, prevention of dental caries, post-eruptive enamel breakdown restorations or extractions and lastly, maintenance.

According to Mathu-Muju and Wright (2006)[99], the following clinical criteria should be considered in an attempt to create clear divisions between the severity levels of MIH:

Mild MIH: "[1]Demarcated opacities which are in non-stress-bearing areas of FPM, [2] isolated opacities, [3] no enamel loss from fracturing present in opaque areas, [4] no history of dental

hypersensitivity, [5] no caries associated with the affected enamel, and [6] incisor involvement is usually mild if present"[100].

Moderate MIH: "[1] intact atypical restorations can be present, [2] demarcated opacities are present on occlusal/incisal third of teeth without post-eruptive enamel breakdown, [3] post-eruptive enamel breakdown/caries are limited to 1 or 2 surfaces without cuspal involvement, [4] dental sensitivity is generally reported as normal, [5] aesthetic concerns are frequently expressed by the patient or parent"[100].

Severe MIH: "[1] posteruptive enamel breakdown is present and frequently occurs as the tooth is emerging, [2] there is a history of dental sensitivity, [3] often widespread caries is associated with the affected enamel, [4] crown destruction can readily advance to involve the dental pulp, [5] defective atypical restoration is present, [6] aesthetic concerns are expressed by the patient or parent"[100].

Prevention

Appropriate diet and prevention advice should be given to patients with MIH and their parents[101]. Some preventative advices should include: Use toothpaste with a higher fluoride levels, with at least 1000ppm fluoride concentration; use topical varnish e.g. Duraphat 22600 ppm F, this may help in reducing sensitivity and may assist in the processes of the remineralaisation of the hypomineralised areas[65].

Casein Phosphopepetide-Amorphous Calcium Phosphate (CPP-ACP) might seal and act as a source of calcium and phosphate for MIH teeth[101,20].

Using 0.4% stannous fluoride gels on a daily basis also seems to be helpful for reducing sensitivity in defective teeth[102].

Fissure sealants (FS) may be used for mild defects but the use of them should be monitored regularly[19,99,102]. Compared to conventional FS, FS appeared to have greater retention when applied using 5th generation adhesive prior[99,103]. Mathu and Wright (2006)[99] suggested a 60-second pre-treatment with 5% sodium hypochlorite if the fissures appeared opaque or yellow-brown. This works to potentially remove intrinsic enamel proteins. Glass Ionomer Cements (GIC) can be used as FS and the benefits of doing so are plenty. Firstly, it provides temporary protection against caries, it improves sensitivity protection, and it minimizes break-downs; as retention is poor, these GICs should be replaced as soon as the tooth is fully erupted with resin-based sealants[19]. The only available research for FS is a recent prospective clinical study regarding MIH molars with occlusal opacities[103], the study suggests that FS seems to have greater retentive capabilities when applied using adhesive prior to sealant.

Restoration of MIH

Two experimental approaches have been proposed to prevent MIH: The first approach is the removal of all defective enamel until sound surfaces are reached[19,99]. This approach would require removal of substantial tooth substance; but it will provide sound enamel for bonding. The second approach to restore MIH involves removal of the porous enamel, until resistance to the probe or to the bur is felt[102,104]. This approach is less invasive, but it may result in defective enamel continuing to breaks down.

There are many restorative materials available to the dentist treating patients with MIH: Glass Ionomer Cement (GIC), Resin Modified Glass Ionomer Cements (RMGIC), Polyacid modified composite resins (PMCR), Composite resins (CR), and amalgam.

Amalgam is used in atypically shaped cavities; due it's non adhesive property non supported enamel will further breakdown[19,102,105].

Restorations with GIC, RMGIC and PMCR are not recommended in FPMs and they can only be used as intermediate or temporary approaches until a definite restoration is placed[19,101,106] .

Fayle (2003)[35], Mathu-Muju and Wright (2006)[99], William et al (2006a)[19] and Willmott et al (2008)[101], all agreed upon the great properties of CR, in combination with new adhesive materials presented.

Adhesion to hypomineralised enamel: Fearne et al (2004) [58]and Heijs et al (2007)[107] indicated that the 'full thickness enamel' which surrounds the clinically defective lesions is less affected. They have also highlighted that the underlying dentine of hypomineralised enamel undergoes no major structural changes. These findings may explain the acceptable results for adhesive CR restorations in molars with MIH, if all apparently defective enamel is removed.

Preformed Metal Crowns (PMC) on FPM have been used for many years to restore molars with defective enamel; until this day, they are recommended as a viable treatment option for MIH posterior teeth[19,102,108]. The use of PMCs prevents further loss of tooth, it establishes correct interproximal and proper occlusal contacts, it controls sensitivity, and lastly it is not costly and requires little time to prepare and insert. They also found that there existed no significant differences between the longevity and success rates for PMC and cast adhesive copings (nickel chrome alloy)[35]. It has been suggested that the 'Hall' technique for PMC[109], with entails no tooth reduction, could be used; however, it is important to note that there are no reports on its long-term efficacy and it is possible that greater occlusal problems may occur. A study by Koch and Garcia Godoy (2000) [110]presented good results with regards to using the 'Hall' technique; the problem, however, lies in that they evaluated only a very limited number of aesthetic crowns, as opposed to a much greater sample of gold crowns

Restoring Hypomineralised Permanent Incisors

According to Jälevik and Noren (2000)[9] the yellow or brownish-yellow defects are more porous with full thickness, whilst those that are creamy-yellow or whitish-creamy are less porous and varying in depth and are mainly located in the inner part of the enamel. As a result, these second type of defect mentioned may occasionally respond to bleaching with carbamide peroxide. On the other hand, the use abrasive paste with 18% hydrochloric acid may be effective only in shallow whitish defects. There are a number of side effects associated with the use of bleaching, with 10%-38% carbamide peroxide, to counter brownish-yellow defects, particularly in immature teeth. Some of these side effects include sensitivity, mucosal irritation and enamel surface alterations and increase with higher concentrations of carbamide peroxide[108,111]. As such, the use of bleaching, particularly in immature teeth, is recommended against.

Another approached, namely the "etch-bleach-seal" technique, follows a three-step model and has been suggested by Wright (2002)[112]. According to this conservative approach, the yellow-brown defects are etched with 37% phosphoric acid for 60 seconds, the defects are then bleached with 5% sodium hypochlorite and for 5-10 min, and lastly the defects are covered with a FS over the surface to occlude porosities and to also prevent re-staining. For the following 5 years, acceptable clinical results were reported[113]. Furthermore, if opaque resins are placed and direct CR veneering is applied after any enamel reduction, aesthetic improvements may very well be achieved [102,114]. The pattern, extent, and change in hardness produced are currently unpredictable with regards to the ability of caries infiltrate resin in penetrating MIH enamel lesions[115].

Other options for anterior MIH defective teeth in children as well as in adolescents with larger enamel defects include restorations with CR and veneers[116] . There may be a reduction in long-term positive aesthetic results as veneers which use CR may be susceptible to discolouration,

wearing and marginal fractures[117]. Some studies recommended porcelain veneers for older children and adolescents[116,108].

Extraction and Orthodontics

Conditions affecting the decision of extraction include the child's age, orthodontic considerations, presence of other dental anomalies, pulp involvement, presence of third molar germ(s), degree of severity of MIH, and lastly the degree to which the tooth is restorable and consequently the expected long term treatment cost[118,99]. The dental age of 8.5-9 years is the ideal time for the FPM extraction. A full clinical examination and a panoramic radiograph will be of great assistance in evaluating signs for acceptable results[119]. With a potential to help the eruption of the second molar into a good contact with the second premolar, complete crown formation and the initiation of the calcification of the bifurcation of the permanent second molar is sought after and indeed desired[119].

2.8 Proposed Treatment Decision Plan For MIH Teeth[100]

Ongoing preventive care for all cases

Mild Defects

Identified as enamel opacities without break-down, with no/slight sensitivity, and with mild aesthetic problems, and no caries.

Molars: Fluoride varnish in partially erupted teeth when fully erupted, sealants with prior adhesives. CR restorations if break-down or caries occur. Full porcelain crowns, if needed, in adulthood.

Incisors if needed:

The etch-bleach-seal approach should be used in brownish-yellow defects of younger children, or chair-side bleaching with 10% carbamide peroxide in older.

In whitish defects, microabrasion followed if needed by CR restoration should be the course of action. The CR restorations should follow the enamel reduction.

Moderate/severe Defects

Identified as enamel break-down, atypical restorations, sensitivity, caries, and aesthetic problem.

Molars:

Extraction should be an option in this case. The dentist should apply fluoride varnish or GIC in partially erupted teeth and CR restorations for up to 3 surfaces.

PM crowns or copings for more than 3 surfaces Full porcelain crowns in adulthood.

Incisors if needed:

It is preferable that the dentist waits until the defect gets better, since a degree of enamel mineralisation may occur in the salivary environment.

After micro-abrasion or enamel reduction and intermediate opaque resins, CR restorations may be an option, or veneers.

Porcelain veneers if needed in adulthood.

3. Aims

The aim of this study was to study the prevalence and severity of MIH among school children in (Dubai), UAE. There is no published information on the prevalence of MIH and the severity of MIH in Dubai, UAE. The data generated might act as a platform for the prevalence of MIH in Dubai.

4. Materials and Methods

4.1 Study Design, Location and Population

A cross-sectional study among public school Emirati children in Dubai, United Arab Emirates.

The calculation of the sample size is explained in the below section.

4.1.1 Sample Size

Sample size calculation:

To calculate the sample size and because no previous studies were conducted in the United Arab Emirates, the sample size was based on the findings of a study conducted in Jeddah, Saudi Arabia by Allazzam et al 2014[38].

Number of sample size in thestudy was 267 patients, with prevalence rate of 8.6%

p = Prevalence

B = width of 95% C I

C= Corrected factor: The ratio of the proportion of MIH between community and hospital base

$$N = \frac{z^2 p(1-p)}{B^2} * C$$

The sample size (N) = $\frac{(1.96)^2(0.086)*(0.914)}{(0.02)^2} * \frac{8.6}{17.6}$ = 368.9 ≈ 369 cases.

The Power of the test

Power is the probability of rejection the null hypothesis when it's false (1-β). When we have one sample for estimation of proportion (just like prevalence) we have a hypothesis that the proportion is different from zero, hence we use the following formula

$\Phi = \phi(-z_{1-\alpha} + \sqrt{n}/\text{p(1-p)})$

So the power will be equal to:

1-β = ϕ=ϕ(-1.645+0.01*√396/0.01*0.99)= ϕ(-1.446) = 92.65%

$\frac{342}{369}$×100 = 92.6%

Non-response = 7.4%

As stated if the power of the test exceeds 80% then the test is powerful**.**

These calculations were provided by Dr.Ammar Hassan Khamis (PHD. Biostatics and epidemiology)

4.1.2 Sampling Technique

A random cluster sample was chosen from public schools in the Emirate of Dubai.

First stage: The schools were selected randomly from the school list of the Ministry of Education by using Microsoft Excel 2010.

Second stage: The grades were selected randomly from grade 2 until grade 7.

Third: The students were randomly selected from second stage list.

Consent was given to the randomly selected students and was signed by their parents

4.1.3 Participating Schools

An approval letter was obtained from the Ministry of Health in Dubai and also an approval was obtained from the Dubai Educational Zone to access the sample group in the public schools in Dubai (Appendix I). The following schools were randomly chosen from the list and agreed to participate in the study:

- Jumeirah Model Girls School
- Zayed Bin Sultan Primary School
- Al Maaref School
- Khadijah bint Khowailad Primary School in Hatta
- Hatta Primary School
- Al-Hudaibeiah Primary School

4.2 Inclusion and Exclusion Criteria

4.2.1 Inclusion Criteria

- Children aged 8 to 12 years old.
- UAE nationals.
- Consent for examination given by legal guardians.

4.2.2 Exclusion Criteria

- Children diagnosed with *amelogenesis imperfecta*, tetracycline staining, hypoplasia, diffuse opacities, white spot lesions, erosion, fluorosis, white cuspal and marginal ridges or undergoing orthodontic treatment at the time of the assessment.
- Children who had the consent signed but refused to cooperate or did not have consent for the examination.

4.3 Data Collection

Data was collected using standard form (Appendix VII) through dental examination. The examination was conducted by two principal investigators. An assistant recorded the findings in the data sheet. Initially the data sheet was identifiable by the child's name. Once the data sheet was checked for completeness, the sheets were coded.

4.3.1 Examiners Calibration

The principle investigator was calibrated at Leeds University, UK along with. The calibration was done with the aid of 40 clinical photographs to differentiate MIH from other developmental enamel defects such as fluorosis, *Amelogenesis Imperfecta* and other types of opacities. Both the primary investigator and the data collector were examined separately and then together. Inter-examiner reproducibility was calculated and was found to be good in all parameters examined (kappa=0.89). After 3 weeks using 40 photographs the principle investigator was assessed again and the Intra-examiner (Kappa=0.90).

4.3.2 Dental Examination

Prior to examination all children received instructions on prevention of caries and periodontal disease. The dental exam was performed on a portable dental chair (Figure 1) at the school nursing room. One student at a time was examined using sterile gloves, artificial light, and a disposable mouth mirror[120]. Post examination a letter sent to each child's parent who have been examined ,which included a copy of dental examination results ,information about caries, oral hygiene and MIH ,prevention advise about their oral health status and treatment needs.

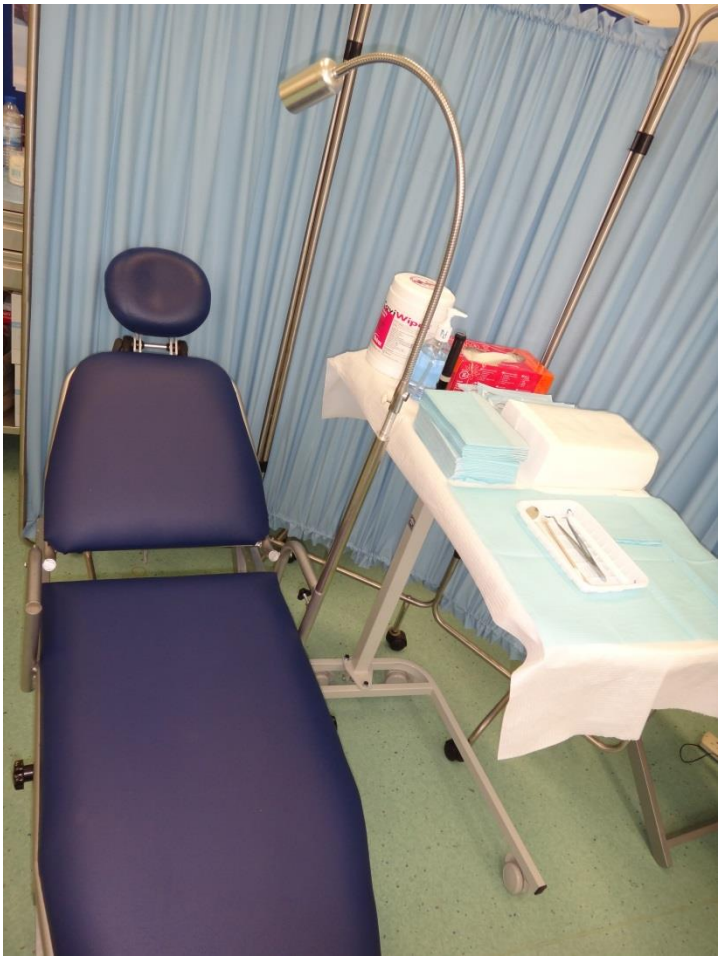

Figure 1: Portable dental chair used to examine the children.

4.3.3 Cross Infection Control

During the examinations, the guidelines of the National Institute for Clinical Excellence (NICE) were followed for cross-infection measures[121].

- Hands were decontaminated immediately before and after examining each patient. This was done by using alcohol hand rubs or hand washing.
- Alcohol hand rubs were used preferably, while liquid soap and water were used if the hands were visibly soiled with bodily fluids.
- Gloves were used as a single-use item for each candidate since there was contact with oral mucosal surfaces and saliva. Gloves were worn immediately before patient contact and removed after completing the examination.
- Natural rubber latex gloves were available as an alternative for patients with a history of latex allergy.
- Gloves were discarded immediately by the examiner into waste disposable bags.
- All instruments were disposable.

4.3.4 Indices

The following indices were used:

A. European Academy of Peadiatric Dentistry (EAPD) criteria for MIH (Molar-Incisor Hypomineralisation). (See Appendix 5)

- This Diagnostic criteria**:**

1. Demarcated opacities. (DO)
2. Post-Eruptive enamel Breakdown. (PEB)
3. Atypical Restoration (AR)
4. Extraction due to MIH

*1. Demarcated opacities: "*The affected teeth show clearly demarcated opacities at the occlusal and buccal part of the crown. The defects vary in color and size. The color can be white, creamy or yellow to brownish. The defect can be negligible or comprise the major part of the crown. It is recommended that defects less than 1 mm are not to be reported[11]"

2. Enamel disintegration (Posteruptive break down): "The degree of porosity of the hypomineralised opaque areas varies. Severely affected enamel subjected to masticatory forces soon breaks down, leading to unprotected dentine and rapid caries development[11]"

3. Atypical restorations: "FPM and incisors with restorations revealing similar extensions as MIH are recommended to be judged as affected[11]"

4. Extraction due to MIH Absence of a molar should be related to the other teeth. Absence of a first permanent molar in a sound dentition is suspected to have been an MIH molar.

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B. Severity index

This index was used to examine the severity status of MIH[100]. The degree of severity and size of the defects were also registered as:

- Severe "loss of enamel with the need for restoration, atypical restoration"
- Moderate "loss of enamel"
- Mild "color change"

The size was recorded as: large (≥4.5 mm), moderate (≈3.5mm), small (≈2mm) respectively[86].

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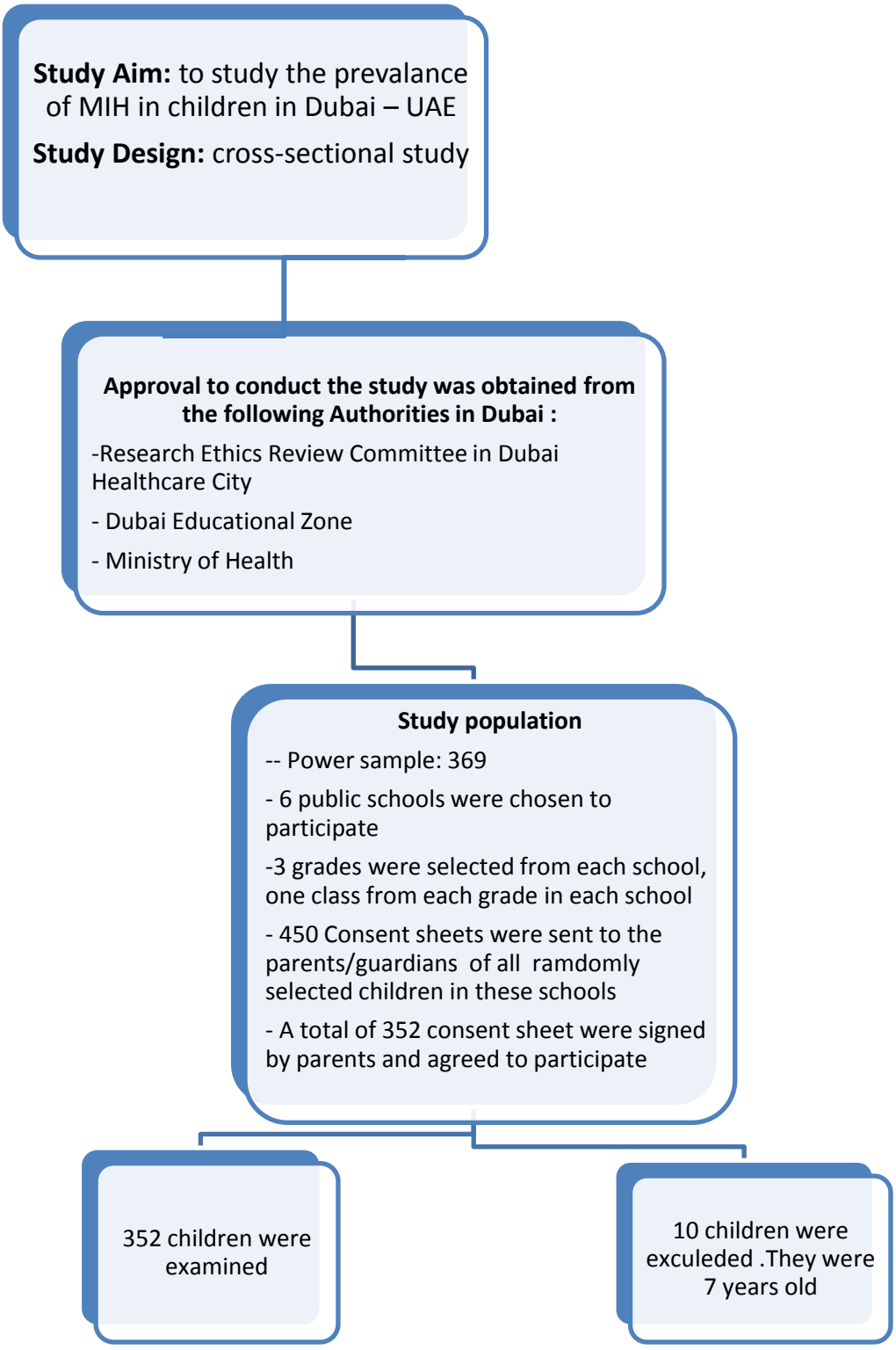

Figure 2: Study methodology summary flowchart.

4.4 Statistical Analysis

Data were entered into a computer using SPSS for Windows Version 20.0 (SPSS Inc., Chicago, IL). Descriptive analysis was used to describe categorical and continuous variables. A cross-tabulated was used to examine the independency between categorical variables and statistical analysis was performed using χ^2-square and Exact Fischer test when appropriate for test of association. P-value of ≤ 0.05 was considered significant in all statistical analysis.

4.5 Ethical Considerations

This study was conducted in full conformance with principles of the "Declaration of Helsinki", Good Clinical Practice (GCP), and within the laws and regulations of the UAE/DHCC. The ethical approval was obtained from the Research Ethics Review Committee in Dubai Healthcare City (Appendix V).

Approval from Ministry of Health and Dubai Educational Zone were obtained to access the public schools.

5. Results

5.1 Study Sample Characteristics

The characteristics of the study sample of children are shown in table 1. A total of 342 children participated in this study: 215 girls (62.9%), and 127 boys (37.1%). In Dubai, 217 children were examined (63.5%) and the remaining 125 children (36.5%) were examined in Hatta. The average age was 9.46, (SD = 1.215).

Table 2: Demographic characteristics of study sample with MIH status

	MIH		
	No	Yes	p-value
Gender			
Male	104(81.9)	23(18.1)	0.002
Female	145(67.4)	70(32.6)	
Age	9.49(1.95)	9.40(1.27)	0.533
Location			
Hatta	86(68.8)	39(31.2)	0.128
Dubai	163(75.1)	54(24.9)	

5.2 Prevalence

The prevalence of MIH was 27.2% (93 out of 342 children). The prevalence of MIH and its different categories is presented in Table 3. There was a statistically significant difference in the proportion of MIH between males and females. Boys had a proportion of 23 (18.1%) while the proportion in girls was 70(32.6%) with a p value of 0.002. No statistically significant difference

in proportion of MIH was related to age (P=0.261). Age distribution of sample of children with and without MIH is shown in Table 3.

In total, 4104 permanent incisors and molars were examined. Of these examined teeth, 196 teeth were affected by MIH. The overall prevalence of MIH among the teeth was estimated as 4.8%.

MH was higher than MIH, 61 out of 93 children (65.6%) had MH while 32 out of 93 children (34.4%) had MIH.

MIH in maxillary permanent first molars were significantly higher (20.8%) than mandibular permanent molars (14.6%) with p= <0.005. Also MIH in permanent maxillary incisors were significantly higher (8.8%) than permanent mandibular incisors (0.9%) with p=<0.001.

The molars most frequently affected by MIH were UR6 (29.9%), UL6 (29.9%), and the least frequently affected tooth was LL6 (20.2%). For the incisors, the upper left central incisor UL1 was the most frequently affected incisor by MIH (11.1%) and the least frequently affected incisor by MIH was the lower left lateral incisor LL2 (0.6%). Figure 1 demonstrates the distribution of MIH in teeth.

The MIH lesion that was found to be the most common in the examined teeth was demarcated opacities (158 teeth). The upper left first molar "UL6" was the most frequently affected tooth with demarcated opacities at (44.1%).

The presentation of atypical restorations was most frequently found in the lower right first molar "LR6", 21 teeth (7.6%). The more severe presentation of post eruptive breakdown was found in 17 teeth and most frequently in the lower right first molar "LR6" (9.8%). The distribution of MIH by category in the 4104 teeth is shown in Table 4. None of the molar teeth screened were extracted. All these findings are illustrated in Figure 2.

Table 3 (below): Prevalence of MIH and different categories among 342 children screened for MIH

Items	Number/total	Prevalence
MIH	93/342	27.2
Demarcated Opacity	85/342	24.9
Posteruptive breakdown	15/342	4.4
Atypical Restoration	14/342	4.1
Extraction	0/342	Zero

Age	MIH		
	No	Yes	
8	69 (70.4)	29(29.6)	0.261
9	52(69.3)	23(30.7)	
10	79(76)	25(24)	
11	35(83.3)	7(16.7)	
12	14(60.9)	9(39.1)	

Table 4 (above): Age distribution of sample of children with and without MIH

Items	Number/total	%
MIH	196/4104	4.8
Demarcated Opacity	158/4104	3.8
Post eruptive breakdown	17/4104	0.41
Atypical Restoration	21/4104	0.52
Extraction	0/4104	Zero

Table 4 (above): The proportion of MIH and different categories in screened teeth

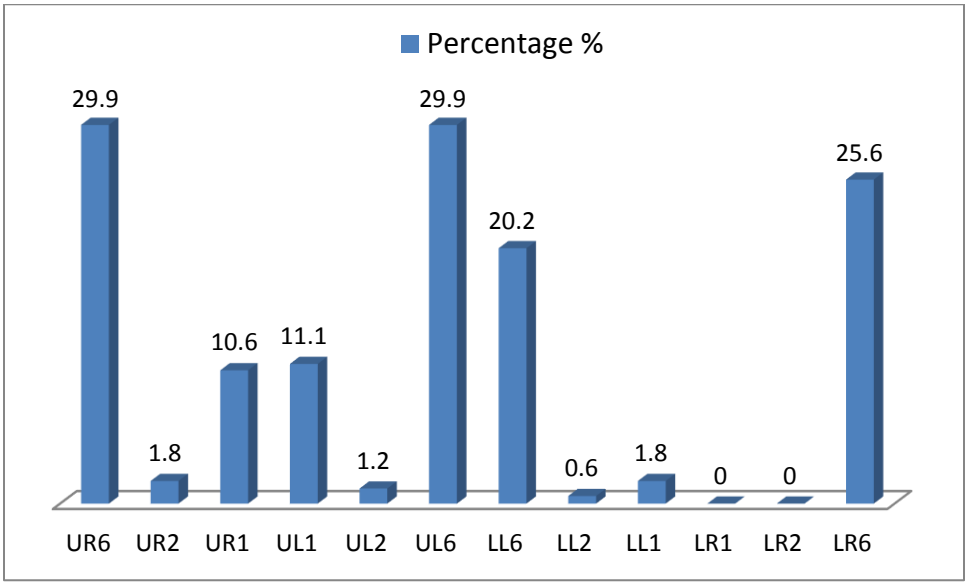

Figure 3 (above): Distribution of the percentage of MIH in affected teeth

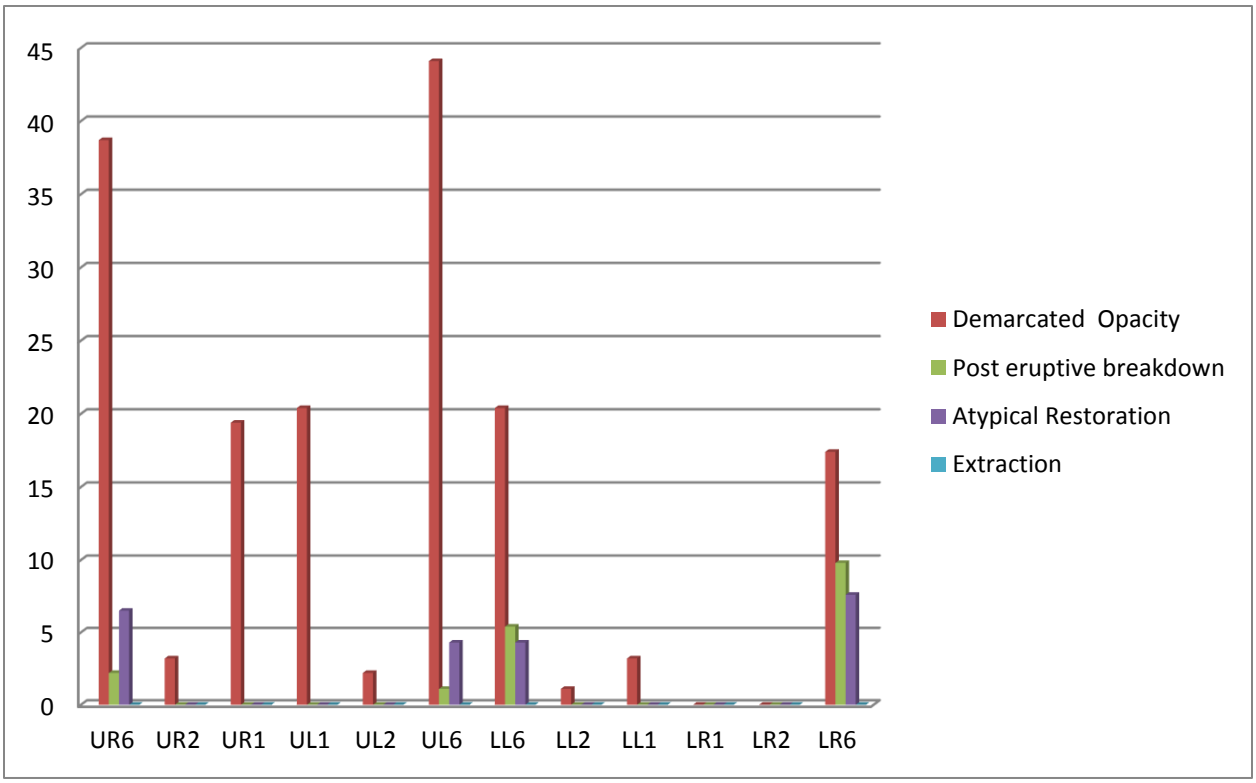

Figure 4 (above): Distribution of criteria of MIH per teeth screened

5.3 Severity

There were variations in the severity levels of MIH. Of the 93 children found to be affected, (53%) presented mild lesions, (17%) presented moderate lesions and (30%) had severe lesions. Figure 5 demonstrates the severity of MIH among children in Dubai.

The molar tooth most frequently affected with severe lesions is the lower right first molar "LR6" 20 (5.8%), while the upper left first molar "UL6" was most frequently affected by moderate lesions 10 (2.9%). The upper right first molar "UR6" and the upper left first molar "UL6" 28 (8.2%) were most frequently affected by mild lesions.

The most frequently affected incisor with severe MIH was the upper right central incisor "UR1" 2 (0.6%). The same tooth, the upper right central incisor "UR1" 3(0.9), was most frequently affected by moderate lesions. The upper left central incisor "UL1" was most frequently affected by mild lesions 17(5.0). Table 6 illustrates the distribution of the severity of MIH per tooth.

Table 5 (below): The severity of MIH per teeth

	UR6	UR2	UR1	UL1	UL2	UL6	LL6	LL2	LL1	LR1	LR2	LL6
None	298 (87.1)	339 (99.1)	324 (94.7)	323 (94.4)	340 (99.4)	295 (86.3)	314 (91.8)	341 (99.7)	337 (98.5)	342 (100)	342 (100)	309 (90.4)
Mild	28(8.2)	3(0.9)	13(3.8)	17(5.0)	1(0.3)	28(8.2)	16 (4.3)	1(0.3)	2(0.6)	0	0	9(92.6)
Moderate	5(1.5)	0	3(0.9)	2(0.6)	1(0.3)	10(2.9)	2(0.6)	0	0	0	0	3(0.9)
Severe	11(3.2)	0	2(0.6)	0	0	8(2.3)	10 (2.9)	0	1(0.3)	0	0	20(5.8)

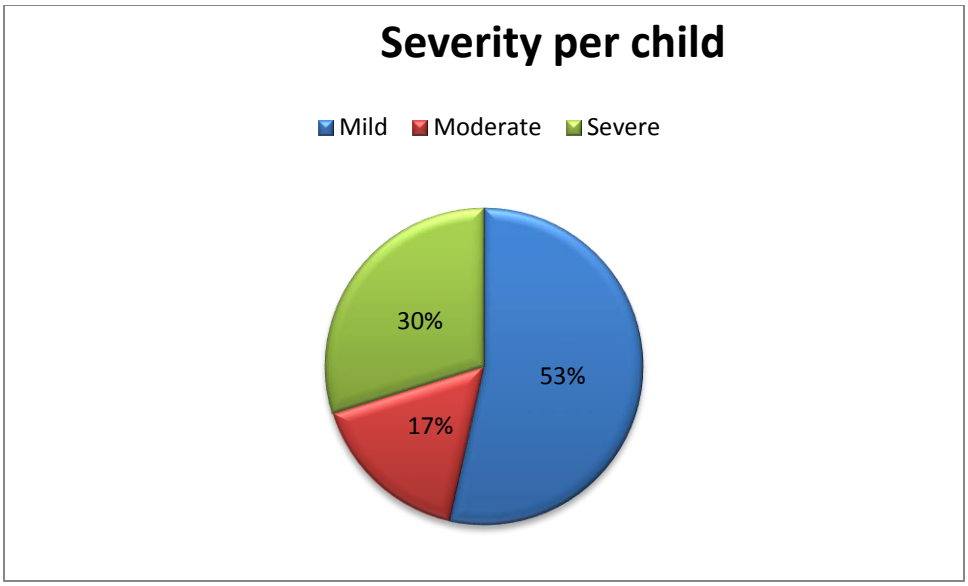

Figure 5 (above): Percentage of severity of MIH among children in Dubai

5.4 Demarcated opacities, Post eruptive breakdown and atypical restoration in Hatta and Dubai

There was an overall significant gender difference in the presence of demarcated opacities; there existed a higher prevalence in females (P=0.002). There was no statistically significant difference in the proportion of demarcated opacities between Dubai and Hatta, 48(22.1%) and37 (29.6) respectively (p-value = 0.08).

There were also no statistically significant differences in post eruptive breakdown and atypical restoration between males and females and also between Hatta and Dubai.

A summary of differences in Demarcated opacities, Post eruptive breakdown and Atypical restoration in Hatta and Dubai is presented in Table 6 a, b and c respectively.

Table 6: Demarcated opacities in Hatta and Dubai

Table 4a	Demarcated Opacity		p-value
	No	Yes	
	Gender		
Male	107(84.3)	20(15.7)	0.002
Female	150(69.8)	65(30.2)	
	Location		
Hatta	88(70.4)	37(29.6)	0.08
Dubai	169(77.9)	48(22.1)	

Table 4b	Post eruptive breakdown		p-value
	No	Yes	
	Gender		
Male	204(94.9)	11(5.1)	0.285
Female	123(96.9)	4(3.1)	
	Location		
Hatta	117(93.6)	8(6.4)	0.135
Dubai	210(96.8)	7(3.2)	

Table 4 c	Atypical Restoration		p-value
	No	Yes	
	Gender		

Male	124(97.6)	3(2.4)	0.169
Female	204(94.9)	11(5.1)	
	Location		
Hatta	123(98.4)	2(1.6)	0.063
Dubai	205(94.5)	12(5.5)	

6. Discussion

An early detection of MIH is crucial as affected teeth frequently display quick post-eruptive enamel loss which in turn results in rapid caries progression and pain. MIH teeth frequently display increased sensitivity which might hinder proper oral hygiene measures for these vulnerable teeth. Despite the importance of early diagnosis and management, the data regarding the prevalence of MIH are lacking from this region of the world. The availability of this data will undoubtedly lead to better knowledge and education about this condition.

6.1 Prevalence

The present study found that the prevalence of MIH in children in Dubai was 27.2%. Our results were slightly higher than the prevalence of MIH in neighbouring countries. The prevalence of MIH in Iraq was found to be 21.5%[122], Iran 20.2%[37], Jordan 17.6%[25] and KSA 8.6%[38]. All of these studies utilized similar criteria for MIH diagnosis (EAPD 2003)[123], however there are some differences in the age groups and the sample sizes. This current study used the EAPD (2003) index[123] excluding children below the age of 8 years to diagnose MIH and a sufficient sample to represent Dubai.

The highest prevalence of MIH yet was reported in Brazil (40%)[43] and in Denmark (38%)[44] . Based on previous observations by dental practitioners in Hatta, a rural area in the Emirate of Dubai, reports of a high prevalence of MIH by many prompted us to investigate the difference in the prevalence between Dubai City and Hatta. There are few reported studies in the literature for intercountry variations of MIH prevalence; in this study the difference in the prevalence of MIH between Dubai and Hatta was investigated and found to be statistically insignificant. Two studies in Brazil reported a higher MIH prevalence in children born and living in rural areas compared to urban areas [23,124]. The authors explained this difference by the presence of better public health

services in urban areas compared to rural areas. Another study conducted in Germany reported that the larger the city is the higher is the MIH prevalence[125]. Although there was no significant difference between the two locations in our study, we noted that in Hatta there was a higher proportion of MIH (31.2%) compare to a proportion of (24.9%) in Dubai. Hatta had a higher proportion of demarcated opacities (29.6%) compared to Dubai (22.2%). As for post eruptive breakdown, Hatta had a prevalence of (6.4%) compared to (3.2%) in Dubai. Regarding atypical restorations, Hatta had a lower prevalence (1.6%) compared to (5.5%) in Dubai. This might be due to better access for dental services in Dubai leading to more restorations. The above findings although not statically significant, might reveal a tendency towards higher prevalence and severity of MIH in Hatta compared to Dubai. The high prevalence of MIH in Dubai could be due the high level of antibiotic use in the UAE, where people can purchase it without any prescription .this might increase the risk of MIH as Whaltling and Fearne (2008)[68] concluded in their study.

It is very important to note the prevalence and severity of MIH in children in order that appropriate preventative measures are taken and more awareness of MIH takes place among the population. Similar to many previous studies[,37,38,125], we reported on the prevalence of specific presentations of MIH in the 93 affected children. Demarcated opacities in FPMs were the most frequent defect type (24.9%) followed by post eruptive breakdown (4.4%) and atypical restorations (4.1%).

6.2 Gender, Age and location

Most of the studies in the literature found no association between prevalence of MIH and gender[3,23,33,36,42,125,126]. Some studies, however, have revealed a higher prevalence of MIH in girls

than in boys which is in line with our present findings[25, 72,127]. In this study, 70 girls had MIH (32.6%) compare to 23 boys (18.1%) with a p-value of 0.002. Some authors have suggested that this may be due to the more advanced eruption age of females compared to males, resulting in discrepancy of MIH prevalence between genders[104]. In this study the number of girls examined (215) was higher than the boys (127). This might have resulted in higher prevalence of MIH in girls.

In this study age was not significantly associated with MIH like some other studies[24,42]. Although there are some studies which reported differences in prevalence of MIH in the examined age span of children[23,127]. An explanation for that might be the fact that MIH occurs during tooth development and is manifested as soon as the tooth is erupted in the mouth. Although the severity of the lesion might progress with age; the prevalence would not.

6.3 MIH and MH (MIH among teeth)

Since MIH is a significant oral health problem in children, it is very important to investigate which teeth have a higher proportion of MIH. The proportion of teeth affected by MIH was lower 196 out of 4104 (4.8%) compare to prevalence of MIH in children 93 out of 342 (27.2%) children. Most of the studies reported the frequency of MIH diagnosis criteria among children but few reported the proportion of MIH among teeth[25,38,128]. Our findings showed that most children are affected, in more than one molar, with MIH- a finding consistent with many other studies[34,126,127]. Children were reported to have higher prevalence of MH[129] compared to MIH in many studies[34,25,125]. In the current study we found that molars were affected far more than incisors, 61 out of 93 cases (65.6%) for molar, while the number of cases for incisors was 32 out of 93 children (34.4%). These finding corroborate several other studies[25,42,127]. This might be due

to the timing of completion of the molar formation and calcification which occurs in molars before incisors during tooth formation.

The proportion of demarcated opacities per tooth was found to be 158 out of 4104 (3.8%), while post eruptive breakdown was found in 17 out of 4104 (0.41%) and atypical restoration was present in 21 out of 4104 (0.52%) . Interestingly enough, none of the children we examined had any first permanent molar extracted or missing. Some studies did not report anything regarding extracted permanent teeth[25,38]; they only reported other criteria for MIH. This could be explained by professional and cultural points of view regarding the extraction of the FPM in children. Many dental professionals in the UAE do not support the extraction of the FPM as a method of treating severe MIH and allowing the second permanent molar to move mesially and close the space; this is according to the guidelines of the enforced extraction of FPM by Royal College of Surgeons of England[130]. There is also a cultural stigma against extraction of permanent teeth in the UAE, regardless of the prognosis of the teeth. Also it could be due to that the average age of the study population a proximally 9 years .

6.4 Differences in Maxillary and Mandibular Molars

MIH in maxillary permanent first molars was significantly higher (20.8%) than mandibular permanent molars (14.6%) with a p-value = <0.005.

The first permanent molars most frequently affected by demarcated opacities were UR6 (38.7%) and UL6 (44.4%). These findings agree with many studies[4,34,131,132] and also disagree with others[5,25,127]. Some studies reported insignificant differences between the arches[2,33]. However, permanent mandibular first molars were more severely affected. In our study, we found out that first mandibular molars had more post eruptive break down, LL6 (9.8%). In addition, LL6 had more atypical restoration than other first permanent molars (7.6 %) this indicated the higher

severity of MIH in these teeth and might indicate different treatment needs and strategies. A possible explanation for this phenomenon is the sequences of eruption, given that mandibular molars erupt before maxillary counterparts[127].

6.5 Incisors

MIH in permanent maxillary incisors was significantly higher (8.8%) than permanent mandibular incisors (0.9%) with a p value <0.001. In this study, children had remarkably increased frequencies of demarcated opacities in incisors, most affected incisors were the UL1 (20.4%) and the least affected incisors were the LL2 (1.1%). These findings are similar to previously reported studies[25,127]. These finding cannot be explained by the sequence of eruption since the lower incisors usually erupt before the uppers. It might, however, be due to the earlier completion of crown formation of the maxillary incisors compared to the mandibular incisors.

6.6 Relationship between opacities and gender

In the present study, girls were found to have higher prevalence of demarcated opacities than boys which is similar to earlier findings [42,44]. However, generally there is agreement amongst authors that no gender differences exist in demarcated lesion prevalence [4,131].Variations in prevalence estimation by gender may be the result of differences between chronological tooth developments between genders. Differences in defect severity between genders may also be attributed to variation in oral care and behaviour[37].

6.7 Severity

It is essential to assess the severity of MIH in addition to the prevalence as this has significant implications on assessing treatment needs. In the current study, the severity of MIH teeth was assessed according to suggested criteria by Lygidakis (2010)[100]. In the present study, a large majority of the children with MIH (53%) presented mild defects and this finding is in agreement with various previously mentioned reports[23,24, 34]. The percentage of moderate defects among MIH children in the present study was 17% and the severe defects percentage was 30% which was higher than the moderate lesions. In some recent studies, a higher prevalence of severe defects was reported[125]. Many studies suggested that the reason for a higher prevalence of severe MIH is related to age. Children with severe defects are at increased risk of enamel loss and breakdown due to occlusal forces[4,23,24,25, 34,42].

6.8 Treatment Needed

Some useful clinical indicators were suggested by a study in Singapore to the clinicians to assess the risk of enamel breakdown in the tooth. The higher the number of MIH teeth, the higher the risk of enamel breakdown[133]. The darker the colour of opacity, the less resistance the tooth is to structural loss, and the more likely the tooth is to develop post-eruptive breakdown and subsequently atypical restorations[23].

Most of the previous studies found that MIH children had higher caries prevalence "DMFT (decayed, missing and filled permanent teeth)" values compare to non-MIH children[4,23,33]. Oftentimes, the MIH teeth suffer from post-eruptive breakdown due to the porosity of the enamel, which leads to exposed dentinal tubules, which are wider in these teeth and cause dentin hypersensitivity. Dentin sensitivity leads to accumulation of dental plaque and as such, the patient will neglect brushing. In addition it can causes severe discomfort and pain for the patient,

which poses an additional challenge for the clinician to provide adequate restoration for relieving the patient's discomfort[20]. Adequate anaesthesia is a challenge to a patient with MIH as well as the dentist; the lack of adequate anaesthesia may result in more anxiety and uncooperativeness on the part of the child[34]. Looking at the child's dental profile and assessing the risk of severe MIH will assist the clinicians to provide a customised prevention and treatment plan for children with MIH. Recommendations include having more frequent review visits, and the need for full coverage restoration prior enamel breakdown and/or planning the timely extraction of these molars[133] .

6.9 Study limitations

The limitations in this current study are as follows:

- The present study showed that MIH is an existing problem in Dubai. However, findings are not representative of the Emirati community as a whole. To better understand the condition in the country, a national oral health survey may be conducted. This study may provide baseline information which can be used for more extensive future research that can involve different regions of the country.

- Since caries is a major concern in children with MIH, it would have been advantageous to include DMFT Index alongside the EAPD 2003 criteria in the examination. However, this was unachievable due to time limitations, number of researchers examining the children and the available facilities to accommodate the large number of participants.

- The study sample was randomly selected from the schools in the Emirate of Dubai. The study sample had a higher number of females than males. An equal number of both

genders might have provided a clearer view of the differences in the prevalence and severity between males and females.

7. Conclusions and Recommendations

- This current study had concluded that the prevalence of MIH in 8- 12 year old children in Dubai is 27.2% with no age predilection.
- The prevalence of MIH was significantly higher in girls 32.6% compare to 18.1% in boys.
- The proportion of children affected with MH (65.6%) was more than children with MIH (34.4%).
- The proportion of MIH affected maxillary first permanent molars (20.8%) were significantly higher compared to mandibular first permeant molars (14.6%).
- The proportion of MIH affected maxillary permanent incisors (8.8%) were significantly higher compared with mandibular permanent incisors (0.9%).
- There was no statistically significant difference in MIH prevalence between Dubai and Hatta (rural area). However, compared to Dubai, there was a higher tendency found in Hatta in with regards to MIH, demarcated opacities and post eruptive breakdown.
- The prevalence of demarcated opacities was significantly higher in girls (30.2%) compared to (15.7%) in boys.
- MIH affected children with Mild defects were 53%, followed by children affected by severe defects (30%) and moderate defects (17%).

Looking at the outcome of this study, the following recommendations are suggested for future research:

- To conduct a similar study including all children in the United Arab Emirates in order to have a better understanding of national oral health and treatment needs.
- To conduct a study with an equal number of both genders to provide a clearer view of the differences in the prevalence and severity of between males and females.

- It would be advantageous to include DMFT Index alongside the EAPD 2003 criteria in the examination to compare caries between MIH children and non MIH children.
- To focus on clinicians and parental awareness programs that stress the importance of diagnosis, prevention programme and treatment needs of MIH in children.

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9. Appendix

Appendix I: Letter of approval from the Ministry of Health – Dubai

Appendix II &III: Study Consent sheets to be signed by parents/legal guardians

Appendix IV: Data sheet

Appendix V: Ethical approval from the Research Ethics Review Committee in Dubai Healthcare City, Dubai, UAE

Appendix I

UNITED ARAB EMIRATES
MINISTRY OF HEALTH

الإمارات العربية المتحدة
وزارة الصحة

التاريخ :2014/04/14م
الرقم : و.ص.د.ط/2014/64
قسم المتابعة والتنسيق

المحترم

السيد/ د. ديفيد راي

عميد وبرفسور طب الفم – كلية دبي لطب الأسنان

تحية طيبة وبعد ،،

الموضوع : الموافقة لإجراء دراسة
حول صحة الفم والأسنان في المدارس الحكومية بإمارة دبي

تهديكم أطيب التمنيات ونتمنى لكم دوام التوفيق والتقدم.

كما أننا نشكر لكم سعيكم في إجراء دراسة للبحث عن مشاكل صحة الفم والأسنان في المدارس الحكومية، وبما أن هذه الدراسة سيكون لها الأثر الإيجابي لمعرفة أسباب ونسبة انتشار مشاكل صحة الفم والأسنان ووضع برنامج طبي متكامل لصحة الفم والأسنان يتضمن الإجراءات العلاجية والوقائية ومساعدة الأهالي على تفهم أهمية الجانب الوقائي وتشجيع أطفالهم على العناية بصحة الفم والأسنان فإنه لا مانع لدينا من إجراء هذه الدراسة على أن يتم تزويد إدارة الصحة المدرسية بنتائج البحث الميداني والإحصائيات وتحليل النتائج للإستفادة منها لوضع لاخطط المستقبلية للصحة المدرسية وخاصة قسم صحة الفم والأسنان.
الرجاء التواصل مع الدكتورة ماجدة – نائب مدير ادارة الصحة المدرسية على هاتف 6770169-050.

شاكرين لكم حسن تعاونكم معنا ،،
وتفضلوا بقبول فائق الاحترام والتقدير ،،،

ناصر خليفه البلوشي
مدير منطقة دبي الطبية بالإنابة

Appendix II

Consent for research:

Research Title: prevalence of MIH in Dubai-U.A.E

Dear parents\guardian:

We are a group of pediatric dentists from Dubai School of Dental Medicine currently studying the prevalence of Molar Incisor Hypominerlaisation (MIH) in children in Dubai in the United Arab Emirates. Recognizing prevalence of MIH in children will help us structure a proper dental program according to their treatment needs. We will be visiting the schools to conduct a simple dental checkup, which is free of charge. You will be provided with a copy of your child's dental examination results. The collected data will be used for research purposes only and your child's information will be kept confidential (The data will be scanned and save in the electronic file dedicated for the research data on Dubai School of Dental Medicine server). This research has minimum risk that not exceeds a simple dental examination to your child. Your child's voluntary participation will serve a great value to the community.

- If you are interested to participate, please sign below and answer the attached questionnaire and will cause no
- Kindly note that only signed forms with complete answers will be eligible to participate.
- Only children who meet the study's inclusion criteria will be included.
- The Child's information will be written in the data sheet and questionnaire for the purpose of collecting data ,after confirmation of completeness of data the name will be coded
- Parents can withdraw their child from the study at any time without any liability on their part, and withdrawal will not affect any treatment they receive.
- The data of the child will be destroyed upon withdrawal from the study.

I have read and understood the above information and AGREE for my child (Name: ----------) to participate.

The data might be used for future research projects provided that your child's identity will not be revealed. Approval by Research Ethics Review Committee in Dubai Healthcare City will be obtained for any future use of your child's information.

☐ Agree
☐ Disagree

For my child (Name: ----------) to participate.

Name _____

Relationship to child _____

Contact number – mobile: _____

Signature _____

For inquires please call: 04-4248624 ,(Dubai College of Dental Medicine)

Or call Dr.Ghada Hussain 052 9797977

This research study has been approved by the Research Ethics Review Committee in Dubai Healthcare City, Dubai, and UAE

Appendix III

عنوان البحث : خلل في تكوين طبقه المينا الخارجيه لاسنان الاطفال مدينة دبي

عزيزي ولي الأمر :

نحن مجموعة أخصائيين (طب أسنان الأطفال)من كليه دبي لطب الأسنان ، نجري دراسه حول الخلل في تكوين طبقة المينا الخارجية لاسنان الأطفال من اماره دبي.

الهدف الأساسي من هذه الدراسة: التعرف على نسبه انتشار هذا النوع من الخلل التي يواجهها الاطفال في مجتمعنا، وكيفية وضع الحلول المناسبة التي تلبي احتياجاتهم العلاجية والوقائية.

كما هو معلوم فإن أي دراسة بحث تستلزم وجود أشخاص ينضمون ويشتركون في الدراسة ،لذا سنقوم بزيارة المدارس وإجراء فحص أسنان مجاني للأطفال وتسليم نتائج الفحص لكل طفل ، علماً بأن هذا الفحص يتضمن الحد الأدنى من المخاطر التي لا تتجاوز فحص الأسنان الدوري ، البيانات التي سيتم رصدها ستستخدم لهذا البحث ،مع الإحتفاظ بسريه تامه للمعلومات الشخصية لطفلك(حيث ان المعلومات سوف تحفظ في الملف الالكتروني الخاص بالبحث الجاري في كلية دبي لطب الاسنان) . علماً بأن لجنة البحوث الطبية وافقت على هذه الدراسة مسبقاً.

مشاركة طفلك في هذه الدراسةهو عمل تطوعي ويعتبر خطوة فعالة نحو مجتمع واعي و صحي.

- اذا كنت ترغب في المشاركة الرجاء التوقيع أدناه و الإجابة على الإستبيان المرفق.
- يرجى العلم بان الاستمارات الموقعة و الاستبيانات الكاملة هي التي ستكون مؤهلة للمشاركة في الدراسة.
- الأطفال المستوفون لشروط البحث سوف يتم ادراجهم في هذه الدراسة.
- معلومات الطفل ستكون مكتوبه على ورقة البيانات والاستبيان لغرض جمع البيانات، بعد التأكد من اكتمال البيانات، اسم الطفل وبياناته ستكون مشفرة
- ولي الامر له الحق باخراج طفله من الدراسة في اي وقت من غير اية تبعات دون ان يؤثر ذلك على علاج المريض
- في حالة انسحاب الطفل من الدراسة سوف يتم التخلص من المعلومات بشكل نهائي.

لقد قرأت وفهمت المعلومات الواردة أعلاه وأوافق على مشاركة طفلي (الاسم: ---------- ------- ----------) في الدراسة البحثية.

يمكن أن تستخدم البيانات للمشاريع البحثية في المستقبل شريطة أن هوية طفلك لن يتم التعرف عليها.

البيانات التي سيتم رصدها ستسخدم للبحوث المسقبليه ،مع الإحتفاظ بسريه تامه للمعلومات الشخصية لطفلك مع العلم بانه ستؤخذ موافقه اخرى من قبل لجنة مراجعة اخلاقيات البحث العلمي في سلطة مدينه دبي الطبية للدراسه المستقبليه .

☐ اوافق
☐ غير موافق

اسم ولي الأمر: التوقيع :

رقم الهاتف المتحرك : التاريخ :

شاكرين لكم تعاونك معنا .....

للا ستفسار الرجاء الاتصال على الرقم:04.4248624 (كلية جبي لطب الاسنان)

او الاتصال د.غادة حسين 0529797977

تمت الموافقة على هذه الدراسة البحثية من قبل لجنة مراجعة أخلاقيات البحوث الطبية في مدينة دبي الطبية، دبي الإمارات العربية المتحدة

Appendix IV

EAPD 2003(Wheerheijm et al 2003)

Name: ----------

Age: ---------

Gender: Female, Male

Location: Hatta , Dubai.

TOOTH #	Sound	UE	DO	PEB	AR	EXT	Severity
16							
12							
11							
21							
22							
26							
36							
32							
31							
41							
42							
46							

DO→Demarcated Opacities

PEB→Post eruptive Enamel Breakdown

AR→Atypical restoration

EXR→Extracted due to MIH.

Severity index:

Mild: less than 1/3 of the tooth, no enamel loss .Demarcated opacities are in non-stress-bearing areas of FPM, there are isolated opacities, no enamel loss from fracturing is present in opaque areas, there is no history of dental hypersensitivity, there are no caries associated with the affected enamel, and incisor involvement is usually mild if present.

Moderate: more than 1/3 tooth and less than 2/3, enamel loss only .Intact atypical restorations can be present, demarcated opacities are present on occlusal/ incisal third of teeth without posteruptive enamel breakdown, posteruptive enamel breakdown/caries are limited to 1 or 2 surfaces without cuspal

involvement, dental sensitivity is generally reported as normal, aesthetic concerns are frequently expressed by the patient or parent

Severe: more than 2/3 of the tooth. Posteruptive enamel breakdown is present and frequently occurs as the tooth is emerging, there is a history of dental sensitivity, often widespread caries is associated with the affected enamel, crown destruction can readily advance to involve the dental pulp, defective atypical restoration is present, aesthetic concerns are expressed by the patient or parent.

Appendix V

حكومة دبي
GOVERNMENT OF DUBAI

سلطة مدينة دبي الطبية
DUBAI HEALTHCARE CITY AUTHORITY

May 5th 2014

Dr. Ghada Hussain
Dental Trainee-Pediatric Dentistry
Dubai School of Dental Medicine
Dubai Healthcare City
Dubai, United Arab Emirates

Subject: Ethical Approval for Research Protocol

Dear Dr. Hussain,

This is with reference to the initial protocol application for the research study entitled, "The Prevalence of Molar Incisor Hypomineralisation in Dubai City in UAE, A Cross Sectional Study" which was submitted to the Dubai Healthcare City Authority-Research Ethics Review Committee (RERC) for review and approval.

It is hereby confirmed that the RERC has reviewed the above application on December 9th 2013 and this was followed by further reviews upon submitting revised protocols. On May 5th 2014, the RERC members have unanimously decided to **approve your final submission that was made on April 1st, 2014.**

Please note however, that this ethical approval is conditional to the following:

1. It is at the discretion of the principal investigator to ensure that all the scientific details and background information contained within the protocol are validated and substantiated with evidence to ensure credibility of the research outcome.
2. Other regulatory approval/s, needed to conduct the study is/are to be obtained and submitted to the RERC for record keeping.
3. No deviations from or changes to the protocol are to be implemented without prior review and documented approval of the RERC.
4. The research study documentation shall be periodically subject to RERC audit.
5. Upon completion of the study, a "Final Research Study Report" will be required for submission to RERC. Consequently, any abstract/publication should also be brought to the attention of the RERC.

Kindly collect your original Ethical Approval Letter from the CPQ Office after midday on Tuesday May 6th 2014.

We congratulate you and wish you continued success in DHCC.

Best Regards,

Laheeb Al-Mutwalli
Director-Licensing Department
Center for Healthcare Planning and Quality
Dubai Healthcare City Authority

مركز التخطيط والجودة للخدمات الطبية
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www.dhcc.ae/cpq
ص.ب: ٥٠٥٠٠١، دبي، الإمارات العربية المتحدة. هاتف: ٠٩٧١٤٣٤٢٥٥٥٥+، فاكس: ٠٩٧١٤٣٦٢٤٧٧٠+، بريد إلكتروني info@cpq.dhcc.ae
P.O. Box: 505001, Dubai, U.A.E, Tel. + 97143425555, Fax + 97143624770, E-mail: info@cpq.dhcc.ae