MOLAR INCISOR HYPOMINERALIZATION

ABSTRACT

MOLAR INCISOR HYPOMINERALIZATION (MIH) is described as hypomineralization of systemic origin that affects one to all the first permanent molars and is often associated with affected permanent incisors. Dental enamel is a highly mineralized tissue of ectodermal origin. Disturbances in initial matrix secretion phase of amelogenesis will result in quantitative or morphologic defect of enamel. Systemic conditions or environmental insults occur during first 3 years of life, are associated with etiology. MIH molars are fragile and caries can develop very easily in those molars. Children often avoid brushing the sensitive molars, and this will aggravate the situation. Treatment may be painful due to difficulties in gaining effective analgesia. The affected teeth often require repeated treatment due to continuous disintegration of the enamel and difficulties with bonding.

Keywords: Molar incisor hypomineralization, Enamel Opacity, Treatment considerations

INTRODUCTION

Molar incisor hypomineralization (MIH) was first noted in Sweden in the late 1970s in view of the chronological distribution of enamel defects. Molar incisor hypomineralization is defined as a hypomineralization of systemic origin that affects one to all of the first permanent molars and is often associated with affected permanent incisors (Weerheijm et al., 2001). Molar incisor hypomineralization can create serious problems for the dentist as well as for the child affected.

ETIOLOGY

The exact nature of the systemic insult is poorly defined, although an increase in childhood illness has been implicated. It is known that enamel formation begins about week 20 in utero for the crowns of the permanent first molars, 3-4 months for the central incisors and lower laterals and 10-12 months for the upper lateral incisors. It is thought to take about 3 years for crown formation to complete. Therefore, research into the aetiology of molar incisor hypomineralization has concentrated on an environmental insult occurring in the first 3 years of life because of the pattern of molars and incisors affected. Ameloblasts are extremely sensitive, and if disturbed during their secretory phase, it results in reduced thickness of normal enamel which is hypoplasia. However, as it is opacities that occur in molar incisor hypomineralization, the ameloblasts must be affected in the later mineralization or maturation phase of amelogenesis.

PREVALENCE

An epidemiological study showed a prevalence of 9.7-19.3% from Denmark, Finland, Netherlands and Sweden. The typical clinical feature of these hypomineralized molars and incisors are demarcated enamel opacities, often followed by breakdown of the enamel. The appearance is asymmetrical, that is, one molar can be severely affected while the contra-lateral molar may be clinically sound or have only minor defects. The same applies to incisors, although enamel breakdown is uncommon in them.

CASE REPORT

A 7 year old female patient, residing in Bhadaj Village, Gandhinagar, reported to the OPD of Pedodontics and Preventive Dentistry in November 2009 with the chief complaint of decay in lower right back tooth since 2 months. Her father had noticed the decay and slightly discolored teeth in the oral cavity and hence came for the treatment. No other family members had similar complains. The child had suffered from upper respiratory tract infections frequently in the childhood
during first 3 years and also taken antibiotics for the
cure of the same.
On general examination, patient was conscious,
cooperative and well oriented to the time, place and
person. No other abnormality was detected in skin,
sclera, nails etc. On extra oral examination face was
symmetrical with mesoprosopic face form and
mesocephalic head form. She had average facial
proportions and normal temporomandibular joint
function.

Intra oral examination of hard tissue revealed that
teeth are in mixed dentition stage. It was found that all
the four permanent first molars were yellow-brown
with demarcated opacities. These opacities were
limited to the cuspal one third of the crown. On
examination, 16 was giving an appearance of cheesy
molar (figure no. 1). 26, 36 showed demarcated
opacities. 46 showed dentinal caries associated with
post eruption breakdown of the involved molar
(figure no. 2). DMFT score was found 3 while deft
(caries index for primary teeth) score was 8.
Mandibular incisors were erupting and not affected.
Maxillary incisors were not yet erupted.

Figure 1: Showing 16 as CHEESY molar appearance &
26 with demarcated opacities.

Figure 2: Showing 46 with Post Eruption Breakdown.

Figure 3: IOPA of 46 showing coronal radiolucency
involving enamel & dentin

Figure 4: 16 restored with S.S. Crown.

Figure 5: 46 restored with Resin composite followed by S.S. crown.
PROVISIONAL DIAGNOSIS
Molar incisor hypomineralization

DIFFERENTIAL DIAGNOSIS
MIH can sometimes be confused with fluorosis or amelogenesis imperfecta. It can be differentiated from fluorosis as its opacities are demarcated, unlike the diffuse opacities that are typical of fluorosis. Also MIH is caries prone and fluorosis is caries resistant.

Amelogenesis imperfecta is often associated with family history and also affects deciduous dentition and only severe cases of MIH mimic the appearance of AI.

INVESTIGATIONS
Intra-oral peri-apical radiograph of 46 (figure no. 3) & 16, showed coronal radiolucency involving enamel and deep dentinal layers indicating dental caries without involving pulp.

FINAL DIAGNOSIS
Final diagnosis of the case was molar incisor hypomineralization affecting four permanent first molars & excluding incisors.

TREATMENT
In the first appointment all caries was excavated in 46 and Dycal® was applied in the dentin to accelerate reparative dentin formation. During subsequent appointment composite restoration was done in 46 to re-build the tooth structure. Caries excavated in 16 and Dycal® was also applied and restored with resin composite. Topical fluoride varnish was applied to reduce the sensitivity and can remineralize enamel, enhance resistance to demineralization by providing a reservoir of fluoride ions for deposition as fluorapatite.

On subsequent visits patient was on regular follow up, after 2 weeks there was no complain of pain hence stainless steel crown was given in 16 (figure no. 4). Stainless steel crown was also given in 46 as a temporary restoration for protection and functioning of molars (figure no. 5). Permanent full cast crown should not be given till complete occlusion is achieved. Preventive resin restoration was done in 36 and 26.

DISCUSSION
Until MIH is a universally recognized condition with an accepted definition and standard diagnostic criteria, any investigation into its aetiology will be compromised to some extent. The term hypoplasia literally refers to a quantitative developmental defect. However, hypoplasia is also commonly and incorrectly used to describe qualitative defects. Causal relationships cannot be assigned definitively because it is studied by relying on parental recall of medical and dental events in their child's first 3 years.

Conditions common in first 3 years such as upper respiratory tract infections, asthma, otitis media, tonsillitis chickenpox, measles and rubella appear to be associated with MIH. The systemic conditions implicated include nutritional deficiency, brain injury, neurological deficiency, nephrotic syndrome, lead poisoning, GI disorders.

Pre-term birth has been associated with increased prevalence of enamel defects. Associations have also been made between presences of polychlorinated di-benzo-p-dioxins (PCDDs) in breast milk.

The complex care involved in treating affected children must address their behavior and anxiety, aiming to provide a durable restoration under pain-free conditions. Restorations in hypomineralized molars appear to fail frequently. There is little evidence-based literature to facilitate clinical decisions on cavity design and material choice.

A 6-step approach to management is described:

1. Risk identification
2. Early diagnosis
3. Remineralization and desensitization
4. Prevention of caries and post eruption breakdown
5. Restorations and extractions
6. Maintenance

Molar incisor hypomineralization's clinical management is challenging due to:

1. The sensitivity and rapid development of dental caries in affected permanent first molars
2. The limited cooperation of a young child
3. Difficulty in achieving anesthesia
4. The repeated marginal breakdown of restorations.

One of the most challenging aspects of the search for better understanding of MIH is likely complexity of its pathogenesis. Basic research is demonstrating that ameloblast are highly susceptible to relatively minor changes in the environment; for example, increases in temperature, hypocalcaemia, and pH levels.
CONCLUSION
The prevalence of MIH appears to be increasing and a common problem for pedodontists. The etiology of MIH is unclear or may in fact, be multifactorial, children born preterm and those with poor general health or systemic conditions in their first 3 years may develop MIH. The early identification of such children will allow monitoring of their permanent molar so that remineralization as preventive measures can be instituted. In the present case likely aetiology is frequent upper respiratory tract infections occurred during the first 3 years of age & antibiotics taken for the cure of same. The complex care involved must address the child’s behavior and anxiety aiming to provide the durable restoration under pain free condition and high risk prevention protocol.

REFERENCES