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Molar Incisor Hypomineralisation in Special Health Care Children—A Review

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ABSTRACT:

Molar Incisor Hypomineralization (MIH) is a qualitative deficiency of enamel that often affects the incisors and can impact anywhere from one to four first permanent molars (FPMs). Weerheijm et al. coined the phrase to describe developmental abnormalities that impact the first permanent molars and permanent incisors. These abnormalities can range from clearly defined, separate, white, yellowish, or brown-colored areas of opacity to significant structural deterioration after eruption. MIH mostly affects the second primary molars, leading to the condition known as Hypomineralized Second Primary Molars (HSPM).The cause of this illness is currently unknown. Nevertheless, research has indicated that a change in the equilibrium of calcium and phosphate or a lack of oxygen supply to ameloblasts results in enamel abnormalities. Several studies have looked into how genes and environmental factors affect the chance of getting MIH. Child born before their due date and children with chronic diseases are more likely to have MIH. There isn't enough information about MIH and HSPM in kids who need extra medical care, and not many studies have been done to add to the world data on the disease. In order to better understand the rates of MIH and HSPM and to help parents and clinicians become more aware of the risks of MIH and HSPM so that they can take the right steps to handle them, this review will give an overview of how common MIH and HSPM are among children with special health care needs.

INTRODUCTION

A developmental, qualitative enamel defect called MIH causes tooth discoloration and fractures due to diminished mineralisation and inorganic enamel components.[1]Originally, this clinical picture affected first permanent molars and incisors.[2]However, similar abnormalities have recently been observed in both primary and permanent teeth of all kinds.[3] Weerheijm *et al*[4] described "developmental defects affecting first permanent molars and permanent incisors, ranging from yellowish, distinct, white, isolated or brown-colored demarcated opacities to severe post eruptive structural breakdown. Shortly after MIH terminology was proposed, the European Academy of Pediatric Dentistry (EAPD) announced criteria specifically aiming at diagnosing and recording MIH". The defect in the enamel's quality is caused by the disruption of ameloblasts during the mineralization and maturation phase of the enamel, which results in the formation of the lesion. Hypoplasia manifests itself as an area of reduced thickness of the enamel in the form of pits, grooves, and bands, whereas the defect that is caused by MIH appears in a white, yellow, or brown color, reflecting the hypomineralized character of the condition.[5,6]

Enamel hypomineralizations in the primary dentition exhibit similarities to those observed in the permanent teeth affected by MIH.[1]Research has found hypomineralizations in the second primary molars, known as hypomineralized second primary molar (HSPM) or deciduous molar hypomineralization. These abnormalities result from enamel mineralization interruptions during second primary molar development. Hypomineralized second primary molars and permanent teeth exhibit similar MIH symptoms.

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HSPM, which is typically linked to MIH, ranges from 0% to 21.8%.[2]

MIH and HSPM have become significant focal points in contemporary pediatric dentistry research. Although there is an abundance of literature on the prevalence of MIH and HSPM among healthy children, there are scant studies on these conditions in children with special healthcare needs. Primary tooth enamel abnormalities are more common in children with cerebral palsy, intellectual impairments, and sensorineural hearing loss.[7] Consequently, this paper aims to compile and review the existing research on the occurrence and prevalence of MIH and HSPM in children with congenital and systemic diseases.

PREVALENCE

The prevalence of MIH has a varied range.

Region	Prevalence
Worldwide	2.4%-40.2%[8]
Europe	14.3%[9]
Asia	13.0%[9]
Africa	10.9%[9]
India	6.31% - 9.46%[10]

MIH was more common in children under 10 (15.1%) than older children (12.1%).[9]Prevalence ranged from 0% to 29.79% among studies.[10]Pooled HSPM prevalence was 6.80%. Preterm children exhibited more enamel developmental defects (69.5% vs 51%) and MIH (38% vs 16%). Low birth weight and gestational age increased MIH risk.[18]Systemically ill and premature children have higher MIH. MIH kids had 12.2% no medical history.[19]

ETIOLOGY

Preterm children have a higher incidence of MIH compared to their full-term counterparts (38% versus 16%), and enamel developmental abnormalities are more prevalent in preterm children (69.5% against 51%). An increased risk of developing mild coronary artery disease was found to be connected with factors such as a low birth weight and a low gestational age.[18] MIH is also more prevalent among preterm children and those with systemic diseases, with only 12.2% of children with MIH having no recorded medical history.[19]

DISCUSSION

Numerous observational studies have been carried out in the years that have followed the establishment of MIH and HSPM as clinical terminology. The purpose of these investigations is to study the possible etiological factors that involve these disorders. In spite of the fact that prenatal, perinatal, or early-life illnesses or experiences have been speculated to be potential factors, a definitive systemic cause has not yet been found. The field has only seen a small number of systematic reviews on this subject, and none of them have been able to come to any solid conclusions. This is mostly due to the fact that there is a lack of evidence, and the difficulty of non-standardized outcome measurements is a significant obstacle. In addition to systemic disorders and general ill health, a number of conditions, including hypertension, maternal diabetes, the use of pharmaceuticals and medications during pregnancy, hypercalcemia, and other types of conditions, have been proposed as potential risk factors for maternal intrauterine hypertension. In addition, MIH has been linked to events such as throat infections, high fevers, and the use of amoxicillin in conjunction with other antibiotics; nevertheless, it is difficult to determine whether or not these factors are primary causes of the condition.[20]

Damage to the enamel organ during amelogenesis can affect tooth enamel.[21]When it comes to primary teeth, amelogenesis starts taking place during the 15th week of pregnancy and continues until the second primary molar is born, which is approximately one year after birth. During the later phases of amelogenesis, disturbances that impact ameloblasts might potentially lead to enamel hypomineralization, which in turn can result in enamel opacities. The process of amelogenesis, which is primarily governed by genetics, can be affected by ambient as well as systemic variables. Enamel hypoplasia is the normal outcome of defects that occur during the secretory phase, whereas enamel hypomineralization is the consequence of defects that occur during the maturation phase. Despite the fact that the etiology of MIH has not been conclusively established, a number of medical disorders, such as prenatal, perinatal, and postnatal diseases, low birth weight, the use of antibiotics, and exposure to chemicals while breastfeeding, have been suggested as possible contributors to the syndrome.[22]Enamel abnormalities can be attributed to a number of factors, including alterations in the calcium-phosphate balance or an inadequate supply of oxygen to ameloblasts. Research has also investigated the impact that genetic and environmental factors play in the occurrence of MIH. It has been observed that the incidence of MIH is higher

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in infants who were born prematurely and in children who had systemic disorders. Only 12.2% of children who have MIH do not have any relevant medical history from their past. Systemic stress during the prenatal or perinatal period, such as a maternal disease, lack of oxygen during birth, as well as medical or environmental insults within the first three years of life (including respiratory and infectious diseases, fever, antibiotic use, and dioxin exposure), have been implicated in increasing the risk for maternal intrauterine hypertension. The mineralization of enamel in a first permanent molar begins shortly before birth and continues until the age of one year. This is the reason why this characteristic occurs. It has also been claimed that MIH is based on a gene-environment model, which suggests that numerous genes, each of which has a tiny influence, are involved. This highlights the necessity for future research to take into account both genotypes and environmental risk factors jointly.[23]

CONCLUSION

For the time being, the etiology of MIH and HSPM is unknown; nevertheless, there is substantial evidence that links them to conditions that occur before, during, and after birth. Due to prenatal, perinatal, and postnatal difficulties, children with MIH have more health issues and compromised teeth than those without the illness.[29]Asthma, pneumonia, otitis media, respiratory tract infections, antibiotics, tonsillitis, breast milk dioxin exposureand juvenile exanthematous fevers are implicated at birth. Despite these links, MIH and HSPM's cause is unknown.[30]

Because of their persistent physical, developmental, behavioral, or emotional issues, children with special healthcare needs need extra care and support. They are more susceptible to enamel abnormalities like MIH and HSPM because to their sensitivity. Thus, children with MIH must be detected immediately to prevent dental cavities, behavior issues, and restorative failure due to the considerable drop in enamel mechanical qualities. Early management prevents caries and MIH-affected teeth from breaking down, reducing dental dread and anxiety in youngsters.

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