The Enigma of Molar Incisor Hypomineralization

Molar Incisor Hypomineralization (MIH) which is currently gaining the attention of researchers worldwide is a qualitative developmental defect of the enamel, attributed to a disrupted ameloblastic function and is frequently associated with the incisors and one or more first permanent molars (FPMs).[1,2] The reported prevalence of this anomaly is 4%–25%.[3] Clinically, the enamel is soft and porous and undergoes breakdown either soon after the eruption or later under masticatory load termed as posteruptive breakdown.[4] A localized and asymmetrical clinical presentation of MIH suggests its concordance with a systemic origin that seems to interfere with amelogenesis, leading to a reduction in hardness and modulus of elasticity of enamel, an increased porosity of 5%–25% compared to normal enamel, an increase in carbon and carbonate concentrations, and an increase in protein content.[3] The condition is becoming a challenge for dentists as the affected teeth are extremely sensitive to even slightest of stimuli such as brushing leading to plaque accumulation and an early onset of dental caries. The presence of subclinical infections and posteruptive breakdown induces an inflammatory reaction that leads to the early involvement of the pulp. This poses a treatment challenge for the dentists as local anesthesia is rendered less effective and children present with an increased anxiety and fear.[5]

A weak evidence exists as regards the etiology of MIH, which has yet to be fully evaluated, though genetic and environmental factors have been proposed.[6] There is no cause-and-effect relationship with the prenatal or perinatal diseases, as well as early childhood illnesses, as findings from the various observational studies cannot be taken at their face value, due to the lack of prospective exposure data and standardized outcome measurement.[7]

The morphological appearance of the hypomineralized enamel of MIH-affected teeth has been investigated by scanning electron microscopy, polarization microscopy, secondary-ion mass spectrometry, and X-ray microscopy. The mineral content of such teeth evaluated using time-of-flight secondary ion mass spectrometry, X-ray microanalysis, and X-ray diffraction has reported a higher carbon content and a reduced calcium: phosphorous ratio of 1.4.[5] The enamel of MIH-affected teeth is overloaded with proteins that hinder the growth of hydroxyapatite crystals by binding with them. Clinically, this increased protein content in such teeth is a major hindrance during etching as their presence compromises the adhesion of restorative materials and the affected molars are 11 times more likely to receive re-treatment in comparison to sound molars. In normal enamel, amelogenin promotes crystal organization and modulates crystal morphology along with ameloblastin and enamelin. These proteins get removed from enamel by proteinases, thus paving way for mineral deposition.

The proteinases responsible for the removal of structural proteins are matrix metalloproteinase 20, and kallikrein-4, the former being predominant during the secretion and early-maturation stage of amelogenesis and the latter during the maturation stage. Farah *et al.*,[8] 2010 using tryptic fingerprint/mass spectrometry (MS/MS) for protein identification of MIH enamel showed that albumin, alpha-1 anti-trypsin, and type-1 collagen were present in abundance in the enamel of such teeth as compared to sound tooth enamel. The effect of surface integrity on the proteomics of MIH-affected FPM using gel electrophoresis tandem MS was studied by Mangum *et al.*,[9] who found that hypomineralized enamel is enriched with nonamelogenin proteins; hemoglobin (12 kDa band) being the major component in broken down MIH enamel lesions and albumin (68 kDa band) being predominant in intact MIH enamel lesions. However, with the continuous development of MS techniques such as matrix-assisted laser desorption ionization coupled MS and liquid chromatography coupled with tandem MS, a broader range of information such as new proteins that are not known till now in MIH-affected FPM and the sequence of peptides can be obtained.

Researchers globally ought to have a sound knowledge of MIH, and they need to plan out structured studies on varied domains for its prevention and management.

Prof. Ashima Goyal

Oral Health Sciences Center, Postgraduate Institute of Medical Education and Research, Chandigarh, India.
E-mail: ashimapgi@yahoo.in

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