Developmental Enamel Defects

Primary teeth. This condition has been described predominantly in developing countries. It is a chronological type of enamel defect signifying a synergistic episode of malnutrition and infection such as acute diarrhea seen as chalky white or yellowish discoloration, which runs parallel to incremental lines of enamel usually seen in the gingival one third of maxillary central and lateral incisors, although lower incisors may be affected occasionally.

Localized enamel defects:

They usually affect the permanent teeth and occur due to chronic periapical infection or trauma to its predecessor. The condition was first described by Turner in 1912 and hence is also called as Turner's Hypoplasia.

Localized enamel hypoplasia can occur when a history of prolonged periapical infection of the primary predecessor was present. Factors which govern the spread of infection from deciduous teeth to their permanent successors are:

- Stage of development of underlying permanent tooth germ: The earlier the infection, the more likely the damage is to be irreversible.
- The length of the time the infection remains untreated: The longer the time, greater is the likelihood of damage.
- Virulence of organisms in deciduous pulp tissue.
- Local and generalized host resistance to infection.

Since crown formation in most teeth is completed by 8 years, most of the damage will take place before this age. Most commonly, the premolars are affected since primary molars are more susceptible to periapical infection. Clinical appearance varies from white/yellow/brown opaque patches to pitting/rough surface elements which on exposure to oral environment undergo yellowish brown discoloration.

Trauma to the primary teeth is another cause of localized enamel hypoplasia especially in upper anterior teeth. Any noted a prevalence of 23% when follow up of 35 primary teeth with a history of trauma was done. Fewer occurrence was seen when the trauma occurred above 4 years of age. Lateral luxation and intrusion cases formed the high risk group while subluxation and extrusion formed the low risk groups. Trauma to the permanent tooth bud during surgical repair of cleft lip and palate, exodontias, jaw fractures, treatment of jaw fractures by osteosynthesis can result in hypoplasia. Clinically, there is a circular yellowish brown discoloration on the labial surface of the maxillary incisor. The tooth germ of permanent maxillary central incisor lies palatally in very close proximity to the apices of roots of its predecessors and hence are most commonly affected. The permanent lateral incisors are located behind the central incisors and hence are protected against labial traumatic forces.

Localized enamel defects may be seen in primary dentition in very low birth weight (VLBW) prematurely born infants. This is due to the pressure exerted by laryngoscopy on the alveoli, during intubation as the infants tend to suffer from respiratory problems during neonatal period. Due to hypoplastic mandible, inadvertent force is often exerted, during intubation. As the force is more on left side, enamel defects usually affect the maxillary incisors on left side.

Localized hypoplasia of primary canine:

Circular hypoplastic defects have been noted on facial surfaces of mandibular canine in primary dentition. Skinner and Hung have proposed it to be due to thinning of labial cortical plates over the tooth bud of canine which predisposes it to injury. Prematurity and maternal diet deficient in vitamin D have been proposed to cause cortical thinning. A higher prevalence is noted in Indo Asian, African-American, Chinese and Japanese populations.

Other factors suggested for enamel defects are perinatal complications, respiratory tract problems, diarrhea, dioxine in human breast milk, vaccines given in early child hood, antibiotics