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THE PREVALENCE OF ENAMEL HYPOPLASIA IN THE CITY OF HYDERABAD: INSIGHTS INTO ITS CAUSES, PATHOGENESIS AND CONSEQUENCES

Dentistry

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ABSTRACT

Objective: To evaluate the prevalence of Enamel Hypoplasia in the city of Hyderabad. **Materials and Methods:** This study was conducted by OroGlee Solutions Private Limited, Hyderabad. A total of 2299 subjects aged between 15 to 50 years were examined for the presence of Enamel Hypoplasia. Out of these, 1631 were males and 668 were females. Oral examination was done using an Intraoral Camera. **Results:** Out of 2299 individuals examined in the study, 32 individuals (1.39%) showed enamel hypoplasia. Of the 1631 males examined, 23 (1.41%) displayed enamel hypoplasia, while among the 668 females, 9 (1.35%) showed enamel hypoplasia. **Conclusion:** Enamel Hypoplasia is one of the most neglected defects because of lack of awareness about it. This study determines the prevalence of enamel hypoplasia and helps to understand its etiology, clinical features and treatment options which are essential for both the public and dental professionals. Timely detection and intervention of enamel hypoplasia hold paramount importance. Due to the scarcity of studies on enamel hypoplasia, this study serves as a crucial resource in enhancing our comprehension of enamel hypoplasia, offering guidance for shaping future research on enamel hypoplasia.

KEYWORDS

Enamel hypoplasia, Neonatal line, Intra oral camera, Prevalence, Hyderabad.

INTRODUCTION:

The human smile, with its array of pearly whites, is a universal symbol of health and vitality. Yet, behind that smile, lies a complex process that can sometimes bear the marks of developmental challenges. Enamel hypoplasia, a condition affecting the outer layer of our teeth, serves as an example of such dental anomalies.

Enamel Hypoplasia, also known as hypo mineralization of enamel, occurs due to the disruption in the secretion of ameloblast cells during the development of teeth. [1] The genetic process of Amelogenesis (formation of enamel) is vulnerable to environmental perturbations. Any abnormality in the process of formation of enamel can result in enamel defects, which cannot be repaired. [2] Clinically, defects in enamel are observed as pits, grooves and generalized loss of surface of enamel. [3] Enamel hypoplasia can develop due to a variety of factors, such as hereditary influences, environmental triggers, nutritional deficiencies, congenital syphilis, hypocalcemia, birth injury, premature birth, excess fluoride ingestion, and idiopathic causes. Additionally, local infections and tooth trauma are known contributors to this condition. [4]

Enamel Hypoplasia leads to various complications which include increased wear, dental sensitivity, greater susceptibility to caries, and poor aesthetics. [3] Depending on the extent of involvement and the severity of the lesions, several therapeutic methods may be used. The primary objective of treating enamel hypoplasia is to restore the structural equilibrium among occlusion, function, and aesthetics, often achieved through methods such as teeth whitening, conservative aesthetic restorations, and enamel micro abrasion. [5,6]

The aim of the present study was to evaluate the prevalence of enamel hypoplasia in the city of Hyderabad, shedding light on its etiology, clinical manifestations, and therapeutic interventions. As there is scarcity of research on enamel hypoplasia in India, the findings of this study will be useful to equip both professionals and individuals alike with the knowledge necessary to know the complexities of enamel hypoplasia effectively.

MATERIALS AND METHODS:

A cross-sectional survey was conducted by OroGlee Solutions Private Limited, Hyderabad, among employees at corporate offices and students from schools in the city of Hyderabad. A total of 2299 individuals were examined at their respective places of work and schools in the city of Hyderabad.

A survey questionnaire was prepared to acquire personal details such as age, gender, habits, relevant dental and medical history. Oral examination was done using an intraoral camera connected to a laptop to record videos of all aspects of teeth. Informed oral consent of the participants was obtained before examination. Approval from the respective school administration was taken to examine their students.

Inclusion Criteria:

Participants in the age group of 15 to 50 years were included in the study.

Exclusion Criteria:

Participants below the age of 15 years and above the age of 50 years were excluded from the study.

RESULTS:

In this study, 2299 individuals aged between 15 to 50 years were examined for the presence of enamel hypoplasia. 32 (1.39%) individuals were found to have varying degrees of enamel hypoplasia. In the total study population, 1631 were males and 668 were females. Out of these, 23 (1.41%) males and 9 (1.35%) females were found to have enamel hypoplasia. (Table 1), (Fig 1)

Table 1: Percentage of individuals with enamel hypoplasia according to gender.

Variables (Gender)	Total Subjects	Subjects with Enamel Hypoplasia (n)	Subjects with Enamel Hypoplasia (%)
Males	1631	23	1.41
Females	668	9	1.35
Total	2299	32	1.39

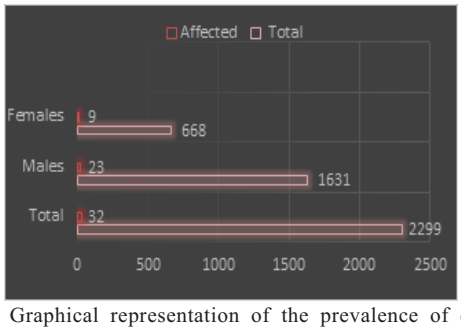

Fig 1: Graphical representation of the prevalence of enamel hypoplasia according to gender

DISCUSSION:

The term enamel hypoplasia refers to a defect in the growth and development of enamel. [3] Enamel defects and abnormalities can be caused by systemic and local factors that disrupt the normal matrix formation. [6]

Enamel is the toughest tissue in the body, which is composed of over 98% minerals and less than 2% organic matrix and water. It is formed through specialized cellular and biochemical pathways. These intricate processes are regulated by genetic factors and can be affected by epigenetic and environmental elements. Any abnormality in the

developmental pathways can lead to a decrease in the quantity of the tissue formed or a deterioration in the quality of mineralization. Ameloblast cells are specialized, end-differentiated cells that form the dental enamel. [7]

ETIOLOGY:

In both primary and permanent dentition, a variety of factors are responsible for enamel abnormalities. Because of the potential for multiple etiologies, the diagnosis of Enamel Hypoplasia must be based on a thorough medical and dental history.

The etiology of enamel hypoplasia can be categorized into two groups: those that result in localized problems restricted to one or a few teeth (such as trauma, infections, ankylosis, and radiation), and those that result in defects affecting most of or all the teeth. Environmental causes or inheritance can lead to such widespread defects. [4]

1. Nutritional Enamel Hypoplasia:

The most common cause of enamel hypoplasia is rickets during the time of formation of teeth. This is due to the deficiency of vitamin D. [8]

2. Enamel Hypoplasia due to congenital syphilis:

The hypoplasia due to congenital syphilis is generally not of the pitting variety but it has a pathognomonic, appearance wherein the upper central incisor is 'screw driver' shaped, the mesial and distal surfaces of the crown taper and converge towards the incisal edge. This hypoplasia involves the maxillary and mandibular permanent incisors followed by first molar. Affected anterior teeth are known as “Hutchinson's Teeth” and molars are known as “Mulberry molars” (also known as Moon's molar, Fournier's molar). [8]

3. Hypoplasia due to Birth injuries:

The neonatal line, introduced by Schour in 1936, is visible in the deciduous teeth and first permanent molars, signifying the transition between prenatal and postnatal enamel formation. It emerges due to sudden changes in the newborn's environment and nutrition. Disruptions during fetal or early newborn stages can impact body functions, including the development of the teeth. Ameloblasts, the cells responsible for enamel formation, gradually recede from the border between dentin and enamel. They incorporate any abnormal tissue formed from disturbances, into the tooth structure permanently. This reflects a type of enamel hypoplasia resulting from disruptions in enamel formation, indicating perinatal trauma or changes in environmental conditions during birth. In cases of traumatic births, enamel formation may halt at this point. Enamel hypoplasia is more prevalent in premature infants compared to full-term babies and is also observed in infants affected by Rh haemolytic disease at birth. [8,9]

4. Hypoplasia due to Local infection or Trauma:

The severity of hypoplasia will depend upon the stage of permanent tooth formation during which the infection occurred, the degree of tissue involvement, and the severity of the infection. When a deciduous tooth becomes carious during the period when the crown of the succeeding permanent tooth is being formed, the bacterial infection involving the periapical tissue of this deciduous tooth may disturb the ameloblastic layer of the permanent tooth, resulting in a hypoplastic crown. Hypoplasia caused by local infection or trauma is unique in that it usually affects only one or a few teeth. The permanent maxillary incisors and maxillary and mandibular premolars are the most common sites. [8]

5. Hypoplasia due to fluoride (mottled enamel):

Hypoplasia resulting from the ingestion of excessive fluoride in drinking water during tooth formation leads to mottled enamel. This type of hypoplasia disturbs ameloblasts during the developmental stage of teeth. Although the exact nature of the injury is not fully understood, histological evidence suggests cell damage, potentially resulting in defective or insufficient enamel matrix production. [8]

6. The primary exogenous contributors include infections, diseases of the haemolytic system, disorders of the heart, kidneys, and gastrointestinal tract, external intoxication, neonatal complications, endocrine disorders and nutritional deficiencies. [10] Idiopathic causes of enamel hypoplasia also include local factors such as infection or trauma to deciduous tooth. [4]

PATHOGENESIS:

Enamel is formed by ameloblast cells. The process of enamel development, known as amelogenesis, starts at the occlusal apex of each tooth crown and moves towards the root and terminates at the cervicoenamel line, which is where the crown and root meet. Stress during this process may cause a transient disruption of ameloblastic activity, resulting in enamel defect, which indicates a cessation of development. [11]

Amelogenesis consists of six stages ¬¬- Desmolytic, Formative, Maturative, Organizing, Morphogenetic, and Protective. Any abnormality taking place during this process, leads to enamel hypoplasia. [4] (Fig 2)

Fig 2: Flow chart depicting the pathogenesis of Enamel Hypoplasia and Clinical features [12] of enamel hypoplasia

CLINICAL FEATURES:

If enamel matrix formation is compromised, Enamel Hypoplasia develops and might show as pitting, grooving or complete absence of enamel. [4]

When maturation is disrupted, hypo mineralization occurs, which is seen as opaque or chalky white patches on usually contoured enamel surfaces. [4] Enamel opacities are round to oval in shape, strongly demarcated, and can be white, yellow, or brown in colour with intact surface. [2]

CLASSIFICATION:

Mild: The surface of the enamel may just have a few tiny pits, grooves, and cracks.
Moderate: The surface of the enamel has horizontal rows of deep pits.
Severe: Enamel formation is significantly affected, resulting in a notable absence of enamel. [6]

Aesthetic Aspects: The manifestations of enamel hypoplasia depend on the etiology and degree of involvement. Horizontal pitting can occur in rows. [6] From minor brownish staining of the enamel to severe pitting and irregularities in the crown (Fig 2). [6]

Cementum, which may be discoloured and brownish-yellow, may be present in hypoplastic defects (it is mostly seen due to local infections or trauma). [6] Enamel Hypoplasia leads to various complications including increased wear, dental sensitivity, greater susceptibility to caries, and poor aesthetics. [3]

Depending on the extent of involvement and the severity of the lesions, several therapeutic methods may be used. These methods often involve teeth whitening, aesthetic conservative restorations, and enamel micro abrasion. With remarkably beautiful results, composite resin restorations are completely capable of mimicking the appearance of a genuine tooth. The central objective in treating enamel hypoplasia is to achieve a balanced interplay between dental occlusion, functional ability, and aesthetic congruity. It also aims to boost patient confidence, which has positive social and psychological effects. [6]

In this study, 2299 individuals aged between 15 to 50 years were examined for the presence of enamel hypoplasia. 32 (1.39%) individuals were found to have varying degrees of enamel hypoplasia. In the total study population, 1631 were males and 668 were females. Out of these 23 (1.41%) males and 9 (1.35%) females were found to have enamel hypoplasia.

Rebecca Slayton et al., conducted a longitudinal study in children who

were part of the Iowa Fluoride Study cohort. This cohort, which has been followed prospectively since birth, involved 698 Caucasian children in Iowa, with the mean age at examination being 4 to 6 years. The prevalence rate of enamel hypoplasia was 6%, and of isolated enamel opacities, was 27%. Teeth most commonly affected with enamel hypoplasia were mandibular second molars (28%), maxillary second molars (25%), mandibular canines (27%) and maxillary canines (23%). Isolated enamel opacities were more common in males than females. [3]

In a study conducted by Alan H. Goodman et al., the prevalence and chronological distribution of enamel hypoplasia were investigated among 300 rural Mexican children aged between 5 to 15 years, who had mild to moderate malnutrition. Enamel defects were identified in 46.7% of the children. Among the fully erupted teeth, the permanent maxillary central incisors exhibited the highest prevalence of enamel defects (44.4%), followed by the permanent maxillary canines (28.0%) and other permanent teeth (ranging from 22.2% to 26.2%). Only 6.1% of the completely erupted deciduous teeth displayed hypoplasia. The distribution of defects across transverse zones indicates a peak occurrence of hypoplasia during the second and third years for permanent teeth. [13]

In a study conducted by Yonezu et al., the prevalence of anomalies in deciduous dentition was investigated among 2,733 Japanese three-year-old children. The findings indicated that enamel hypoplasia was observed in 1.50% of the participants. [14]

A study conducted by Pascoe et al., investigated the prevalence of enamel hypoplasia in 4- to 6-year-old Australian Aboriginal children of the Tiwi tribe on Bathurst Island. The study focused on population groups highly predisposed to this condition, such as those who had serious respiratory tract infections, serious gastrointestinal infections, or anemia during infancy. The findings revealed that 79 out of 80 children (99%) exhibited enamel hypoplasia, with a mean of 12.0 ± 4.1 hypoplastic teeth per child. [15]

CONCLUSION:

Enamel hypoplasia represents a significant challenge in dental health with multifaceted origin including genetic predisposition, environmental factors, and systemic conditions during tooth development. The impact of enamel hypoplasia extends beyond aesthetics to include heightened susceptibility to dental caries, increased tooth sensitivity, and compromised overall oral health. Early diagnosis and intervention are crucial, with treatment strategies ranging from preventive measures like fluoride applications and sealants to restorative procedures such as bonding and crowns. Moreover, the psychological and social implications of enamel hypoplasia cannot be ignored, highlighting the importance of raising awareness among healthcare providers and the general population. By doing so, we can ensure timely intervention and support for affected individuals, addressing both their dental and psychological needs.

CONFLICT OF INTEREST:

There is no conflict of interest.

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REFERENCES:

[1] Erkman, A. C., Go-kart, S. T., & İlbey, S. (2022). Evaluation of linear enamel hypoplasia (LEH) in western Anatolian skeletons from the late eastern roman period (Attepe settlements and Dereköy necropolis). Journal of Archaeological Science: Reports, 41, 103297.
[2] Nota, A., Palumbo, L., Pantaleo, G., Gherlone, E. F., & Tecco, S. (2020). Developmental Enamel Defects (DDE) and their association with oral health, preventive procedures, and children's psychosocial attitudes towards home oral hygiene: A cross-sectional study. International Journal of Environmental Research and Public Health, 17(11), 4025.
[3] Slayton, R. L., Warren, J. J., Kanellis, M. J., Levy, S. M., & Islam, M. (2001). Prevalence of enamel hypoplasia and isolated opacities in the primary dentition. Pediatric dentistry, 23(1), 32-43.
[4] Kanchan, T., Machado, M., Rao, A., Krishan, K., & Garg, A. K. (2015). Enamel hypoplasia and its role in identification of individuals: A review of literature. Indian Journal of Dentistry, 6(2), 99.
[5] Gewehr, A. J. Z. (2012). Aesthetic approach for anterior teeth with enamel hypoplasia. Contemporary Clinical Dentistry.
[6] Ghom, A. G., & Ghom, S. A. (Eds.). (2014). Teeth anomalies. Textbook of oral medicine. JP Medical Ltd., 3rd edition, 105,106
[7] Seow, W. K. (2014). Developmental defects of enamel and dentine: challenges for basic science research and clinical management. Australian dental journal, 59, 143-154.
[8] Sivapathasundharam, B., & Preethi, S. (2020). Odontogenic tumors. Shafer's Textbook of Oral Pathology e-book, 8th edition, 48-52.
[9] Eli, I., Sarnat, H., & Talmi, E. (1989). Effect of the birth process on the neonatal line in primary tooth enamel. Pediatr Dent, 11(3), 220-223.
[10] Krishnaji Musale, P., Shrikant Soni, A., & Sunil Kothare, S. (2019). Etiology and considerations of developmental enamel defects in children: a narrative review. Journal of Pediatrics Review, 7(3), 141-150.
[11] Nikita, E. (2016). Osteoarchaeology: A guide to the macroscopic study of human skeletal remains. Academic Press.
[12] Lacruz, R. S., Habelitz, S., Wright, J. T., & Paine, M. L. (2017). Dental enamel formation and implications for oral health and disease. Physiological reviews, 97(3), 939-993.
[13] Goodman, A. H., Allen, L. H., Hernandez, G. P., Amador, A., Arriola, L. V., Chávez, A., & Pelto, G. H. (1987). Prevalence and age at development of enamel hypoplasias in Mexican children. American Journal of Physical Anthropology, 72(1), 7-19.
[14] Yonezu, T., Hayashi, Y., Sasaki, J., & Machida, Y. (1997). Prevalence of congenital dental anomalies of the deciduous dentition in Japanese children. The Bulletin of Tokyo Dental College, 38(1), 27-32.
[15] Pascoe, L., & Kim Seow, W. (1994). Enamel hypoplasia and dental caries in Australian aboriginal children: prevalence and correlation between the two diseases. Pediatric Dentistry, 16, 193-193.