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Behind the Needle: Can Local Anesthesia Induce Enamel Hypoplasia? A Retrospective Study on Young Permanent Teeth

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Abstract

Introduction: Dental treatment involves similar or more frequent use of local anesthesia compared with any other clinical discipline. Relevant literature- which reported that local anesthetics could accumulate in natural cavities such as the crypts of tooth buds and the mandibular canal. A recent clinical epidemiological study showed that local anesthesia can potentially interferes with human permanent tooth development and induces tooth agenesis through unknown mechanisms. Autophagy is a catabolic process involving the degradation of unnecessary or aberrant cellular components through hydrolysis of lysosomes. It therefore controls the turnover of organelles and proteins within cells, and of cells within organisms

Aim: This study aimed to determine the retrospective effect of Local Anesthesia Exposure on enamel of young permanent tooth

Materials and Method: This is a retrospective study design using a sample of 137 children aged 11-14 years. Children who undergone any dental procedure to their teeth under LA. During the developmental stage permanent 2nd molar tooth was included in this study. Children with other developmental defect like Amelogenesis imperfecta etc were excluded from this study. Total population is divided in to 3 groups, Group A: children with enamel defect in young tooth who does

not undergone any dental procedure under LA, B: Children with enamel defect who undergone treatment under LA in primary dentition period, Grp C: Children without enamel defect who undergone treatment under LA in primary dentition. Data was collected by clinical examination and history taking

Result: Data was analyzed using the statistical package SPSS 26.0 (SPSS Inc., Chicago, IL) and level of significance was set at $p<0.05$. There was a significant association between the age at which the procedure was performed and the presence of enamel hypoplasia

Conclusion: Within the limitations of this study, LA exposure during the developmental stage of tooth may be a predisposing factor for hypoplasia in young permanent tooth, based on the age and site as well as amount of LA solutions

Keywords: Local Anesthesia, Autophagy, Enamel Hypoplasia, Young Permanent Tooth

Introduction

The epithelial compartment of the developing tooth is called the enamel organ and comprises specific cell types, including inner and outer enamel epithelial cells. The inner enamel epithelial cells differentiate into ameloblasts, which go through presecretory, secretory, transition, and maturation stages. At the presecretory stage, enamel formation begins with an organic protein-enriched matrix deposited on dentin by secretory ameloblasts. During the secretory stage, polarized tall columnar-shaped ameloblasts secrete enamel proteins such as amelogenin, ameloblastin, and enamelin. Once the primary enamel is fully thickened, the secretory ameloblasts enter the transition stage, followed by the maturation stage, in which ameloblasts remove protein and deposit minerals in the enamel matrix. Any failure or abnormality in these steps of amelogenesis results in developmental enamel defects. [1]

Local anesthetics are widely used in dental clinics. However, the side effects of this category of drugs have been rarely studied. A recent epidemiological study showed that local anesthetics could potentially induce tooth agenesis. Using detailed dynamic cellular energetic analysis, some studies suggest that these drugs are able to rapidly induce autophagy in the tooth pulp cells, both in animal models and in cultured human cells. The induced autophagy is due to increased mitochondrial respiration, which is believed to counteract the toxicity of the drugs, that is, a protection mechanism. However, this protective machinery failed to function after a longer treatment at high dose, suggesting mitochondrial functions have been possibly damaged[2] Local anesthetics are known to work by binding to voltage-gated Na+ channels in nerves, therefore block sodium transportation and nerve conduction.[1] Although the maximum doses of various local anesthetics are established, the side effects of these agents on dental tissues have not yet been fully investigated. The only relevant literature in this regard relates to a canine model, which reported that local anesthetics could accumulate in natural cavities such as the crypts of tooth buds and the mandibular canal.[2]

Autophagy is a catabolic process involving the degradation of unnecessary or aberrant cellular components through hydrolysis of lysosomes. It therefore controls the turnover of organelles and proteins within cells, and of cells within organisms. During this process, targeted cytoplasmic constituents are isolated within autophagosomes, which then fuse with lysosomes to form autolysosomes where the cellular material is degraded or recycled. It was previously observed that anesthesia drugs could induce vacuolation. However, neither the mechanisms responsible for vacuolation nor its consequence has been reported. Vacuoles have a major role in autophagy and maintain a balance between

biogenesis (production) and degradation (or turnover) for many substances and cellular structures which may interfere with tooth formation and developmental defect in the tooth.[3]

Materials & Methods

This retrospective cohort study investigated the impact of early childhood local anaesthesia (LA) exposure on the enamel development of permanent maxillary second molars. The study was conducted at Department of Pediatric Dentistry Kerala, India, following approval from the Institutional Ethics Committee. Written informed consent was obtained from the parents or legal guardians of all participants, and verbal assent was obtained from the adolescents before enrollment. Total sample of 137 adolescents aged 12 to 14 years was selected for this study.

Inclusion Criteria: Adolescents aged 12–14 years with fully or partially erupted permanent maxillary second molars (teeth #17 and #27), who had verifiable dental records from early childhood (ages 3–7 years), and whose parents provided consent.

Exclusion Criteria: Patients with missing or unerupted maxillary second permanent molars; a history of systemic childhood illnesses known to cause generalized enamel defects (e.g., severe malnutrition, high chronic fevers); genetic conditions affecting enamel (e.g., Amelogenesis Imperfecta); severe dental fluorosis; or those with extensive restorations/crowns on the target teeth.

A retrospective review of the participants' pediatric dental charts was conducted to extract data regarding early childhood dental treatments. History taking via parental interviews was utilized to supplement missing chart data. The independent variable recorded was LA exposure in the maxillary posterior quadrants during early childhood, specifically detailing-Administration of LA for dental procedures (e.g., extractions of primary maxillary molars, pulpotomies, or deep restorations) during the developmental window of the second molar crown. The approximate age of the child at the time of LA exposure. History of periapical infection or trauma to the primary maxillary molars, which served as potential confounding factors.

Clinical Examination

Intraoral clinical examinations were performed by a single calibrated examiner under standard dental chair illumination using a sterile mouth mirror and probe. The maxillary right (#17) and left (#27) second permanent molars were isolated, cleansed, and dried with cotton rolls prior to inspection. The presence of enamel hypoplasia on these specific teeth was visually and tactually assessed. Enamel hypoplasia was defined as a quantitative defect presenting as localized pits, grooves, or partial/complete loss of clinical enamel. The defects were categorized and recorded based on the Developmental Defects of Enamel (DDE) Index.

Statistical Analysis

Data was analyzed using the statistical package SPSS 26.0 (SPSS Inc., Chicago, IL) and level of significance was set at $p<0.05$. Descriptive statistics was performed to assess the proportion of the respective groups. Inferential statistics to find out the association was done by Chi square test.

Results

The total sample consisted of 137 individuals. Most participants (56.9%) had undergone at least one dental procedure in the upper posterior teeth under local anesthesia, while 43.1% had not. (Table-1) Most children who underwent surgery did so at ages 4 (20.4%), 5 (16.8%), and 6 (14.6%), with smaller proportions at ages 7 (2.2%) and 8 (2.9%) (Table 2). There was a statistically significant association between undergoing a dental procedure under local anesthesia and the presence of

enamel hypoplasia (χ^2 = 9.09, p = 0.0001). Among those who had no procedure, 54.7% showed hypoplasia, whereas 45.3% of those who underwent a procedure had hypoplasia (table 3&diagram 3).

There was a significant association between the age at which the procedure was performed and the presence of enamel hypoplasia (χ^2 = 22.13, p = 0.0001). Hypoplasia was highest among those with no procedure (54.7%), followed by children who underwent treatment at ages 4 (20.0%) and 6 (17.3%). Notably, no hypoplasia was observed in those treated at ages 7 or 8. (table 4 & diagram 4)

Table 1: Underwent any dental procedure in upper posterior teeth under LA

		Frequency	Percent
Underwent any procedure?	No	59	43.1
	Yes	78	56.9
	Total	137	100.0

Table 2: Age at which underwent surgery

		Frequency	Percent
Age at which underwent surgery	4	28	20.4
	5	23	16.8
	6	20	14.6
	7	3	2.2
	8	4	2.9
	Nil	59	43.1
	Total	137	100.0

Table 3: Association between LA procedure vs Hypoplasia

			tooth with enamel hypoplasia		Total
			No	Yes	
Underwent any dental procedure in upper posterior teeth under LA?	No	Count	18	41	59
		%	29.0%	54.7%	43.1%
	Yes	Count	44	34	78
		%	71.0%	45.3%	56.9%
Total		Count	62	75	137
		%	100.0%	100.0%	100.0%
Chi square test			9.09		
P Value			0.0001*		

Graph 1: Association between LA procedure vs Hypoplasia

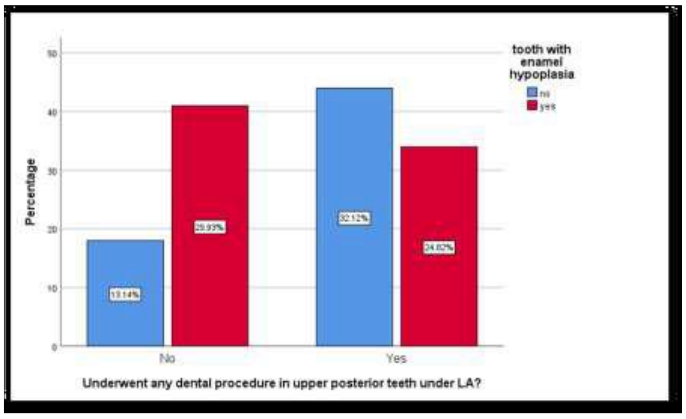
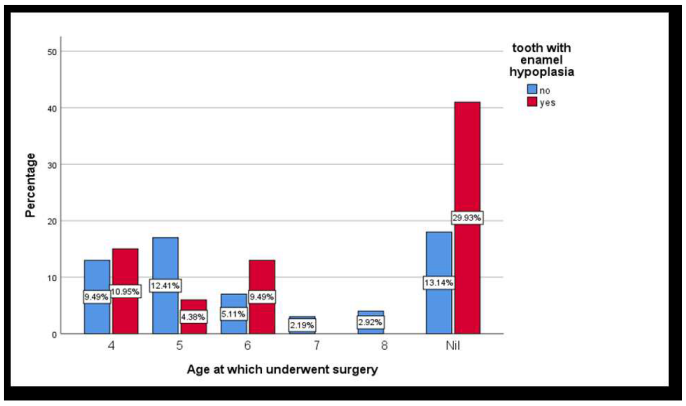


Table 4: Association between Age at which underwent procedure vs Hypoplasia

			tooth with enamel hypoplasia		Total
			No	Yes	
Age at which underwent surgery	4	Count	13	15	28
		%	21.0%	20.0%	20.4%
	5	Count	17	6	23
		%	27.4%	8.0%	16.8%
	6	Count	7	13	20
		%	11.3%	17.3%	14.6%
	7	Count	3	0	3
		%	4.8%	0.0%	2.2%
	8	Count	4	0	4
		%	6.5%	0.0%	2.9%
	Nil	Count	18	41	59
		%	29.0%	54.7%	43.1%
Total		Count	62	75	137
		%	100.0%	100.0%	100.0%
Chi square test			22.13		
P Value			0.0001*		

Graph 2: Association between Age at which underwent procedure vs Hypoplasia



Discussion

The primary objective of this retrospective study was to investigate the impact of early childhood local anesthesia (LA) exposure on the development of enamel in permanent maxillary second molars. Our statistical

analysis revealed a highly significant association between undergoing a dental procedure under LA and the presence of enamel hypoplasia. However, contrary to the conventional hypothesis that LA administration might mechanically or chemically disrupt amelogenesis, our data demonstrated a reverse trend. Specifically, a higher percentage of individuals who did not undergo a dental procedure under LA exhibited enamel hypoplasia (54.7%) compared to those who did undergo a procedure (45.3%).

Crucially, this timeline-dependent pathophysiology is further substantiated by the highly significant association observed between the exact age at which the pediatric dental procedure was performed and the presence of developmental defects Hypoplasia rates were highest among children who never received procedural intervention (54.7%), followed by those treated at age 4 (20.0%) and age 6 (17.3%). Remarkably, no enamel hypoplasia (0.0%) was observed in children who underwent dental treatments under LA at ages 7 or 8.

Teeth development is genetically regulated but can be also sensitive to the action of systemic and/or local acquired risk factors. The acquired DDE occur in the form as hypoplasia, which is a quantitative defect or hypomineralization, in direct relation to the moment when the disturbance takes place. Therefore, hypoplasia appears when the risk factors act during the secretory phase of amelogenesis, while hypomineralizations are caused by the aggressions occurring during the maturation stage of the tooth enamel [4].

There are very few studies on the impact of LA administration during the developmental stage of teeth and the defect on the tooth. In a similar study the authors examined the possible association between IANBs and missing third molars. The authors found a statistically significant greater incidence of missing third-molar follicles in mandibular quadrants that had a definitive history of receiving IANBs compared with mandibular quadrants that had no history of receiving IANB.

In our study those who did not undergo any dental procedure in the past shows 54.7 % of teeth with enamel hypoplasia. A critical methodological and clinical consideration in interpreting these data is the baseline oral health status of the non-procedure cohort. In a standard clinical paradigm, an absence of dental intervention typically implies a healthy, caries-free primary dentition. Ameloblasts are exceptionally sensitive to metabolic disturbances during the active secretory and maturation phases of amelogenesis.[5] If this cohort required no local anesthesia or restorative intervention due to a naturally low caries index, the observed enamel hypoplasia likely stems from early childhood systemic conditions that occurred between birth and age four [6]. Environmental stressors—such as recurrent high pediatric fevers, severe exanthematous viral infections, vitamin D deficiencies, or early childhood malnutrition—exert a systemic cytotoxic effect on ameloblasts. Because the permanent maxillary second molar undergoes intense early mineralisation during this identical chronological window, these systemic disturbances can temporarily halt enamel matrix deposition [5]. Consequently, the primary dentition may remain completely sound and cavity-free (explaining the total lack of procedural history), while the underlying permanent second molars develop deep, structural hypoplastic scars across their crowns.[7]

Effect of computerized delivery of intraligamental injection in primary molars on their corresponding permanent tooth buds was assessed in a study. They concluded that in the primary dentition, C-CLAD-ILI does not increase the danger of developmental disturbances to the underlying permanent dental bud.[8]

Autophagy is fundamentally a "double-edged sword" in dental tissues. At lower, clinical doses, LA-induced autophagy may function as a protective mechanism. In vitro toxicity often relies on sustained, high concentrations of local anesthetics. In a clinical setting, rapid vascular clearance and tissue dilution of the anesthetic agent may prevent the intracellular accumulation necessary to cause toxic, hypoplasia inducing autophagic failure.[2]

Conclusion

Within the limitations of this study, we can conclude that LA cannot be definitively labeled as a direct cause of enamel hypoplasia. Instead, because LA triggers cellular stress and autophagy, it may act as a predisposing factor depending heavily upon the specific site and the concentration of the solution administered. Further studies with a larger sample size are needed to confirm these findings and isolate the thresholds of LA-induced damage.

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