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ORIGINAL ARTICLE

Prevalence of enamel development defects in very low birth weight premature infants

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Abstract

Prematurity has been identified as a leading cause of enamel defects since the 1930s. These defects range from color modifications to more severe dental anomalies. Despite the long-standing recognition of this correlation, there is limited contemporary data on the prevalence of Developmental Enamel Defects (DED) in the primary dentition of children born with very low birth weight (VLBW). This study aims to assess the prevalence of DED in the primary dentition of children aged 12 to 60 months who were born with VLBW. A cross-sectional study was conducted with 93 children followed at a reference university centers outpatient clinic for preterm infants. An intraoral physical examination was performed by a single examiner using the Modified Development Defects of Enamel Index. The prevalence (95% CI) of DDE was 67% (57 to 76), with enamel hypoplasia being the most frequent defect, predominantly affecting the upper incisors. The findings of this study highlight the significant prevalence of DED in children born with VLBW, underscoring the need for targeted dental care and preventive measures in this high-risk group. Early identification and intervention are crucial to mitigate the long-term oral health challenges faced by these children.

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INTRODUCTION

Prematurity, defined as birth occurring before 37 weeks of gestation, is a serious and increasing health issue worldwide. Data from the World Health Organization indicate that the prevalence of preterm births globally ranges from 5% to 18%. From 2011 to 2019, Brazil recorded approximately 3 million preterm births, corresponding to a prevalence of 11%, placing the country among the top ten with the highest rates of preterm births in the world[1].

Premature and low birth weight (LBW, <2500g) infants are very susceptible to all the complications of premature birth both in the immediate neonatal period and during childhood[2] and are particularly prone to oral pathologies, whose are aggravated by neonatal complications like defects on development and growth of the orofacial region, including primary and permanent teeth and developmental enamel defects (DDE) such as enamel hypoplasia, enamel opacity and molar-incisor hypomineralisation. These issues are more prevalent in preterm, specially those <32 weeks or very low birth weight infants (VLBW; <1500g), with enamel defects occurring up to three times more frequently compared to full-term infants[3]. The prevalence of DDE in the primary dentition has been reported at rates ranging between 13% and 45%, and can reach 46% to 96% in children born preterm[4].

Enamel formation begins at 4 months of gestation and continues until 11 months after birth, making it susceptible to prenatal, neonatal and postnatal conditions[5]. DDE are characterized by irregularities in the quality and quantity of dental enamel, resulting from disturbances or damage to the enamel organ. The way these defects present and their severity generally depend on the developmental stage at which the disruption occurs, as well as the severity and duration of the disturbance[6]. Enamel hypoplasia refers to a quantitative defect characterized by enamel deficiency, whereas enamel hypomineralization represents a qualitative defect that involves diffuse, demarcated, or white, yellow, or brown discoloration with increasing severity as the color darkens[5].

These infants miss out on the period of greatest mineral accretion that occurs during the last trimester of pregnancy and are at higher risk of enamel defects. Almost 80% of fetal calcium and phosphorus are accumulated during the third trimester of pregnancy, and significant amounts of minerals are accreted in dental tissue during intrauterine life, explaining the frequent mineral deficiency in premature infants. These deficiencies are exacerbated by rapid depletion due to metabolic demands from conditions like metabolic bone disease, sepsis, persistent ductus arteriosus and peri-intraventricular hemorrhage, which are common in premature infants[7].

The etiology of DDE remains not fully elucidated. Potentially associated factors include socioeconomic status, tobacco use, hypertension, gestational diabetes, use of anti-epileptic drugs during pregnancy, gastrointestinal hookworm infection, and alcohol consumption. A brazilian study published in 2023 identified a reduction in serum levels of the chemokine RANTES in pregnant women between 22 and 25 weeks of gestation as a factor explaining DDE in the primary dentition. This finding suggests that maternal immune response could be a significant factor in the development of DDE in the deciduous teeth, underscoring the need for improved management of maternal health during pregnancy to mitigate enamel developmental defects[8].

A recent 36-month longitudinal study involving 287 LBW preterm children and 290 full-term controls found that LBW preterm children had a 3 times higher incidence of molar-incisor hypomineralization (MIH) and enamel hypomineralization (EH) compared to the control group. Notably, among the 37 VLBW infants included, the incidence of EH and MIH was significantly higher (86.5% and 70.3% respectively) than in full-term controls (26.9% and 21.7%, respectively). These findings highlight the increased vulnerability of LBW and VLBW preterm children to enamel defects, underscoring the need for focused preventive and therapeutic interventions[9]. Additionally, a study by Feuser et al. (2021)[10] involving 84 children aged two to five years showed that LBW children had significantly more teeth with DED compared to the full-term/normal birth weight children, with no significant difference in dental caries prevalence. Admission to the neonatal intensive care unit was significantly associated with the presence of DED, emphasizing the critical need for specialized care in this high-risk population.

DDE have significant clinical implications, they are linked to issues such as aesthetic concerns, dental hypersensitivity, and an increased risk of caries development. Recognizing hypomineralization and hypoplasias is crucial, as individuals affected by these conditions need preventive measures and early intervention in life[11]. This study aims to assess the prevalence of enamel defects in the deciduous teeth of VLBW premature children aged 12 to 60 months.

MATERIALS AND METHODS

This cross-sectional study evaluates premature infants discharged from neonatal intensive care units (NICUs) and subsequently followed in an outpatient clinic in Caxias do Sul between may 2019 and december 2020. Eligible participants were children aged 12 to 60 months, with a birth weight of less than 1500 g and gestational age of less than 34 weeks. The exclusion criteria included genetic syndromes, complex congenital malformations, prior dental interventions, missing teeth, hereditary dental conditions such as amelogenesis and dentinogenesis imperfecta and dental fluorosis.

Outcome and Variables

The primary outcome, developmental enamel defects, was assessed using the Modified Development Defects of Enamel Index as proposed by the International Dental

Federation in 1992. Additional demographic variables were collected including gestational age, birth weight, duration of mechanical ventilation, neonatal sepsis, and metabolic bone disease. Maternal information such as age, smoking status, education level, and prenatal comorbidities was also recorded, alongside post-discharge data, including the age at first tooth eruption and oral hygiene practices.

Data Collection

Informed consent was obtained from the caregivers of all participants. A thorough intraoral dental examination was performed on each child by a single trained observer under artificial lighting conditions, utilizing a wooden spatula and appropriate personal protective equipment. Plaque and food residues were removed as necessary with sterilized gauze. The evaluation of the oral cavity was documented with photographic records for subsequent analysis of diagnostic consistency. Agreement between diagnoses was confirmed through a high level of concordance (kappa = 0.7 [0.59 to 0.80]) with two external pediatric dentists. Each child was included in the study only once, even if they had more than one appointment.

Statistical Analysis

The sample size was calculated based on the described prevalence of DED ranging from 52% to 96% in very low birth weight children. To achieve a study power capable of detecting a prevalence estimate of 65% with a 95% confidence interval (CI) and an absolute 10% margin of error, the required sample size was determined to be 88 participants. Descriptive analyses were performed to determine the prevalence of DED and the distribution of specific defect types. Categorical data were expressed as frequencies, while ordinal and numerical data were presented as medians and interquartile ranges. Associations between qualitative variables were examined using Chi-square or Fisher's exact test, with statistical significance set at 5% (p<0.05). Comparisons of means were made using the Student's t-test, and comparisons of medians were made using the Mann-Whitney U test; Multivariate logistic regression was used to develop a prediction model, incorporating variables with p<0.2 and using backward elimination for variable selection. The model's calibration was assessed using the Hosmer-Lemeshow test and the C-statistic. Odds ratios (OR) with 95% CI were calculated for potential predictor variables. Data analysis was conducted using R software, version 3.5.2.

Ethical Considerations

The study protocol was approved by the Research Ethics Committee of the University of Caxias do Sul (protocol number 08128019.0.0000.5341) and was conducted in accordance with the ethical standards of the 1975 Helsinki Declaration, as revised in 2013.

RESULTS

During the study period, a total of 97 children met the initial eligibility criteria. Exclusions were applied to one child with Trisomy 21, one who had undergone restorative dental treatment, and two without any erupted teeth, resulting in a final population of 93 participants. The clinical and demographic characteristics of the study population are detailed in Tables 1 and 2.

DED was observed in 62 of the children, accounting for a prevalence of 67% (95% CI: 57% to 76%). Significant differences between the group with DED and the group without this diagnosis were noted in the duration of gastric tube use (p = 0.02) and the age at the time of consultation (p <0.01).

The initial univariate logistic regression analysis identified potential associations between the occurrence of DED and two factors: the presence of late-onset sepsis and the extended duration of tube use. However, these associations did not retain their statistical significance upon adjustment in the multivariate model, which included additional variables such as sex, gestational age, duration of tracheal intubation, duration of gastric tube use, and late-onset sepsis (Table 3).

Hypoplasia emerged as the most common defect, predominantly affecting the incisors, followed by canines and molars. Incisors were also most frequently impacted by demarcated opacity, followed by molars and canines. In contrast, diffuse opacity was most prevalent in molars, then canines and incisors. Analysis revealed that incisors, followed by molars, were most commonly afflicted when demarcated opacity and hypoplasia co-occurred. The pairing of demarcated and diffuse opacity was mainly seen in molars, followed bycanines, with the rarest defect being diffuse opacity associated with hypoplasia, observed in canines and then molars. When examining dental arches, the upper arch showed a higher incidence of DED, with incisors, molars, and canines affected in descending order. Conversely, the lower arch's least affected teeth were canines, followed by molars and incisors. The distribution of DED in this population can be seen in Table 4.

DISCUSSION

This study examining the prevalence of DED within a cohort of 93 children, born prematurely and with very low birth weights, revealed three principal findings: a DED prevalence of 67%, a pronounced tendency for DED to affect upper incisor teeth and enamel hypoplasia as the most frequently occurring alteration.

The observed prevalence rate of DED at 67% is consistent with the range of prevalence rates reported in existing literature for premature infants[12-16]. Contrary to findings from other studies that have identified an inverse relationship between gestational age and the prevalence of DED[17], this correlation was not evident in our investigation.

Table 1. Preterm infants' characteristics at birth and maternal characteristics in patients with and without DED.

	DED n= 62 [Q1-Q3]	Without DED n= 31 [Q1-Q3]	*p value*
Male sex	31 (33.5)	17 (18.5)	0.82 #
Birth Weight (g)	1115 [881.2-1258.7]	1230 [962.5-1332.5]	0.05 §
Gestational Age (weeks)	29 [23-33]	29 [23-34]	0.58 §
Apgar Score 5"	8 [8-9]	8 [7-9]	0.65 e
Hospital stay (days)	55 [41.0-75.7]	45 [39.0-68.5]	0.32 §
Intubation (days)	4 [0.0-16.75]	2 [0.0-7.5]	0.24 e
Nasogastric Tube (days)	43 [31.5-56.5]	32.5 [28.0-41.25]	0.02 §
Total parenteral nutrition (days)	15 [10.25-24.5]	11 [9.5-14.5]	0.07 §
Phosphorus (mg/dL)	4,5 [3.27-6.22]	4,95 [4.2-6.62]	0.34 §
Alkaline phosphatase (UI)	429 [332-687]	407 [252.5-458.0]	0.11 §
Sepsis	46 (50)	16 (17)	0.05 #
Bronchopulmonary dysplasia	37 (40)	22 (24)	0.40 #
Exclusive breastfeeding	45 (48)	23 (25)	1 #
Prenatal Consultations	5 [0-13]	6 [0-17]	0.08 e
Mother's age (years)	27,5 [12-40]	26 [14-37]	0.41 §
Maternal Smoking	10 (11)	2 (2)	0.32*
Maternal Diabetes	3 (3)	4 (4)	0.33*
Maternal Hypertension	25 (27)	14 (15)	0.82 #
Maternal Urinary Tract Infection	23 (25)	16 (18)	0.26 #
Maternal Scholarly < 8 years	19 (21)	8 (9)	0.81 #

Legend: *DED = Developmental Enamel Defects; #Chi-square test; *Fisher's exact test; §Student's t-test; &Mann-Whitney U test.

Table 2. Children' characteristics at the time of follow up and its relation with enamel defects.

	DED n= 62 (%) [Q1-Q3]	Without DED n= 31 (%) [Q1-Q3]	*p value*
Age (months)	23 [18.57-35.02]	16.4 [12.55-23.05]	<0.01
First tooth eruption (months)	9 [8-12]	9.5 [8.25-11]	0.95
Oral trauma (months)	17 [13-19]	18 [16.75-18.75]	0.80
Breastfeeding weaning (months)	7 [6-9]	7 [6-8]	0.47
Oral Hygiene (%)	50 (53)	20 (21)	0.15
Previous dentist consultation (%)	12 (13)	7 (7)	0.93

Table 3. Crude and adjusted analysis for covariates potentially associated with developmental enamel defects.

	Univariate model		Multivariate Model	
	OU (IC 95%)	*P* (Wald)	Adjusted OR (IC 95%)	*P* (Wald)
Intubation	2.5 (0.81; 7.73)	0.15	3.25 (0.68; 15.65)	0.14
Sepsis	3.99 (1.25; 12.72)	0.02	3.57 (0.84; 15.21)	0.08
Nasogastric Tube	1.05 (1.01; 1.09)	0.02	1.03 (0.99; 1.08)	0.10
Extreme prematurity	1.50 (0.36; 6.27)	0.85	0.76 (0.12; 4.66)	0.76
Male sex	1.03 (0.36; 3.0)	0.66	0.73 (0.22; 2.47)	0.61

This discrepancy may be attributed to our study's exclusive concentration on VLBW infants, without including comparative groups of infants with higher birth weights. Furthermore, the detection of DED predominantly on both sides of the upper incisors contributes new perspectives on the underlying causes of these defects. It implies that systemic factors, rather than local traumatic events often linked with orotracheal intubation, play a significant role in the development of DED[3,18-19].

The prominence of enamel hypoplasia is particularly noteworthy. The early intrauterine onset of incisor calcification provides a temporal window vulnerable to nutritional and metabolic disturbances[20,21]. The critical role of adequate mineralization, underscored by calcium, phosphorus, fluoride, vitamins A, C, D, and protein, in preventing DED highlights the potential for prenatal nutritional interventions to mitigate risk[22-24].

Despite our rigorous methodological approach, certain limitations were encountered. The retrospective nature of some collected data and the prioritization of prevalence description over analytical evaluation in our sample size calculation may have influenced our findings. The high prevalence of DED observed underscores the urgent need for

Table 4. Distribution of Enamel Development Defects in a Population of Children with a History of Very Low Birth Weight.

	Upper Arch Teeth									
	Molar 2	Molar 1	Canine	Incisors				Canine	Molar 1	Molar 2
Tooth characteristics n (%)	55	54	53	52	51	61	62	63	64	65
Normal	20(88)	44(69)	30(68)	30(41)	22(29)	25(31)	30(41)	24(56)	43(68)	20(88)
Demarcated Opacity	0(0)	4(6)	3(7)	3(4)	6(8)	7(9)	2(3)	5(12)	4(6)	0(0)
Diffuse Opacity	1(4)	6(9)	2(5)	0(0)	0(0)	0(0)	0(0)	1(2)	6(10)	1(4)
Hypoplasia	0(0)	4(6)	5(11)	31(43)	39(51)	41(50)	36(49)	8(19)	3(5)	0(0)
Demarcated and Diffuse Opacity	1(4)	2(3)	1(2)	0(0)	0(0)	0(0)	0(0)	1(2)	2(3)	1(4)
Demarcated Opacity and Hypoplasia	0(0)	1(2)	0(0)	9(12)	9(12)	8(10)	5(7)	0(0)	2(3)	0(0)
Diffuse Opacity and Hypoplasia	1(4)	3(5)	3(7)	0(0)	0(0)	0(0)	0(0)	4(9)	3(5)	1(4)
	Lower Arch Teeth									
	Molar 2	Molar 1	Canine	Incisors				Canine	Molar 1	Molar 2
Tooth characteristics n (%)	85	84	83	82	81	71	72	73	74	75
Normal	20(88)	52(91)	39(93)	66(97)	85(95)	84(95)	65(97)	38(91)	50(89)	21(88)
Demarcated Opacity	0(0)	1(2)	0(0)	0(0)	2(2)	2(2)	0(0)	1(2)	2(3)	0(0)
Diffuse Opacity	1(4)	2(3)	0(0)	1(1,5)	0(0)	0(0)	1(1,5)	0(0)	2(3)	1(4)
Hypoplasia	0(0)	1(2)	1(2)	1(1,5)	3(3)	3(3)	1(1,5)	1(2)	2(3)	0(0)
Demarcated and Diffuse Opacity	2(8)	1(2)	2(5)	0(0)	0(0)	0(0)	0(0)	2(5)	1(2)	2(8)
Demarcated Opacity and Hypoplasia	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
Diffuse Opacity and Hypoplasia	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)

targeted preventive strategies in this vulnerable population. Alongside the epidemiological contribution of our study, the parallel initiation of educational programs for parents and guardians, and the facilitation of access to pediatric dental care represent pivotal steps toward mitigating the oral health risks faced by VLBW premature infants.

The findings from this study reinforce the significant burden of DED in preterm VLBW infants. The presence of DED is likely to increase the risks of developing dental caries, heightened dentin sensitivity, and malocclusions. If left untreated, these conditions would not only impair the oral health but also the overall well-being and quality of life of the affected children. Early intervention, informed by both clinical and educational measures, is thus imperative for promoting the health of this population.

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