Enamel Defects in the Primary Dentition of Preterm and Full Term Children

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Aim: *This study compared enamel defects in children born prematurely and at term.* ***Method****: 96 children born at term (G1), and with 96 children born prematurely (G2) were studied.* ***Results****: A higher prevalence of enamel defects was found in the premature group, with a predominance of hypoplasia. In G1, 64 teeth displayed enamel defects (51 opacities and 13 hypoplasias). In G2, 110 defective teeth were found, (29 opacities and 81 hypoplasias). A significant correlation was found between very low birth weight (VLBW) and the presence of these defects (p≤0.001). The teeth most affected were the incisors, canines and molars.* ***Conclusion****: Prematurity, in conjunction with other factors, can predispose children to enamel defects.*
Keywords: *Dental Enamel; Dental Enamel Hypoplasia; Infant, Premature; Tooth, Deciduous, primary.*

INTRODUCTION

According to World Health Organization, newborns are considered to be preterm when gestation lasts fewer than 37 weeks.[1] Gestational age and birth weight are the main factors determining the incidence of neonatal complications.[2] These complications often require mechanical ventilation and parenteral nutrition which may, in combination with other factors, account for changes in dental development. Recent research has shown that gestational age and intrauterine growth retardation (IUGR), in addition to lack of breastfeeding, increase the probability of alterations in enamel development up to 5%.[3]

Among these changes, developmental defects in enamel are the most extensively researched. These defects are deviations from the normal appearance of tooth enamel, resulting from enamel organ dysfunction.[4] They are found in both primary and permanent teeth,[4,5] and can be classified as enamel opacities and hypoplasia.[4]

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Enamel opacity is defined as a defect involving an alteration in the translucency of the enamel, variable in degree. The defective enamel is of normal thickness with a smooth surface and can be white, cream, yellow or brown in color. The lesions vary in extent, position on the tooth surface and distribution in the mouth.[4] Hypoplasia is a defect involving the surface of the enamel and associated with a reduced localized thickness of enamel. It can occur in the form of pits or grooves and can appear with partial or complete absence of enamel over a considerable area of dentine.[4]

The teeth most affected are incisors, canines and first molars. The second molars are involved less frequently.[5]

The lower the child's weight, the more likely is the occurrence of tooth enamel abnormalities. In children with very low birth weight (<1500 g), approximately 70% have dental enamel defects; and, in the low birth weight group (1500 and 2000 g), the frequency is approximately 40%.[6]

Bilateral hypoplasia of the enamel affects premature infants whenever intrauterine disorders disrupt the amelogenesis process.[7,8,9,10] The prevalence of this alteration in preterm children ranges from 20% to 100%.[3] However, unilateral incisal hypoplasia may be due to traumatic intubation.[7,8,9,10]

It was reported that children born preterm have a higher prevalence of enamel defects in the primary dentition than children born at full term.[2,5,6] This prevalence varies greatly according to study criteria, indices and study sample.

The exact mechanism and etiological factors underlying these enamel defects are not fully understood. Some investigations report that the main etiological factors in metabolic bone disease in preterm births are inadequate calcium and phosphorus supply, and that breast milk contains too little calcium and phosphorus to enable near optimum intra-uterine mineral retention in preterm infants.[5,12,13]

The appearance of structural defects of the enamel, either opacity or hypoplasia, is similar, independent of the causal factor. The FDI, World Dental Federation in 1992, defined the index for structural defects of the enamel for use in epidemiological studies (DDE index). In addition, this publication revealed that the possible causal relation of defects must be established by questioning the parents regarding pregnancy, birth and childhood diseases, to collect

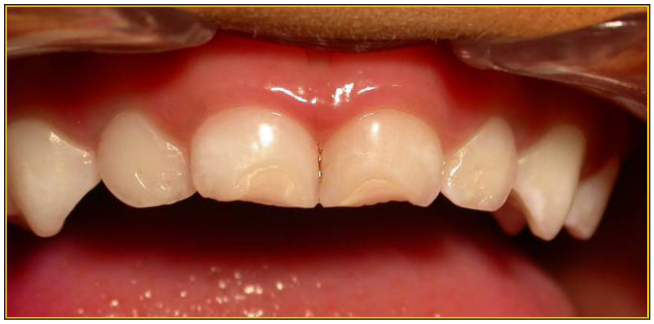

Figure 1. bilateral hypoplasia

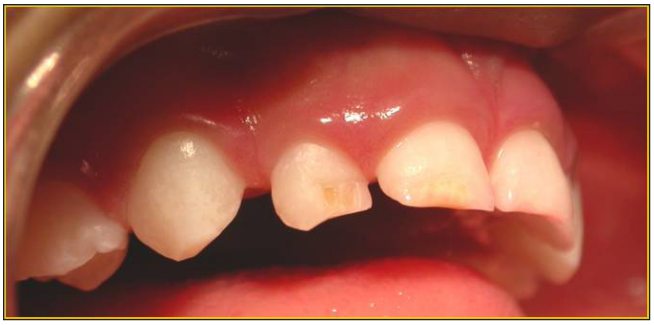

Figure 2. unilateral hypoplasia

data associated with the period of the formation of alterations, according to the odontogenesis.[4] Cases of children with hypoplasias can be seen in Figures 1 and 2.

Very low birth weight and extremely low birth weight were indicators of enamel defects and contributed to an increase in non-nutritive suction habits. These are necessary to establish early preventive and interceptive measures and to avoid future severe problems.[2]

These enamel defects can affect esthetics and can alter occlusion. Furthermore, the absence of enamel can increase the risks for developing dental caries.[12] Therefore, in light of the likelihood of a high prevalence of tooth enamel alterations in preterm children, the purpose of this study was to make a comparison between the enamel defects and related factors present in these children and in a control group of children born at full term.

MATERIAL AND METHOD

This was a case-controlled study with 192 children - 96 born at full term (G1) and 96 at preterm (G2). Children with complete or incomplete primary dentition were included. They were examined at the pediatric neonatology outpatient facility of the Regional Asa Sul Hospital (HRAS), in Brasília, DF, Brazil.

This research was approved by the Ethics in Research Committee of the Federal District Government's Health Secretariat. (Opinion no. 005/2004).

Parents' Questionnaire

After informed consent had been signed by the parents, a questionnaire to record name, age, gender, date of birth and parents' occupation was applied.

Clinical Examination

The examination was performed by a single examiner and the teeth were evaluated for the presence of enamel defects. The defects were analyzed and recorded in a file according to the classification proposed in the DDE Index (1992).[4] The teeth were examined by quadrant for enamel hypoplasia and opacities. The number of teeth with defects was recorded, as well as the total number of erupted teeth. The extent, type and color of the defects were recorded. Whenever hypoplasia and opacity were found on the same tooth, the defect was recorded as hypoplasia. After cleaning and drying the tooth surfaces with gauze, the examination was conducted under natural light, using a dental mirror and explorer probe, with the child lying on a hospital gurney.

The same examiner, calibrated in a previous pilot study, performed the evaluations. To establish the degree of intra-examiner agreement, the Kappa index was used in 10% of the sample, in accordance with World Health Organization (WHO) guidelines. Subjects were reexamined one week after the initial examination (Kappa=0.843).

Gestation and Childbirth Data

Data on gestational period, undercurrent events, medication use, weight and size at birth, APGAR score, neonatal complications, therapies (medications, ventilatory support and parenteral nutrition), length of hospital stay and vitamin supplements were gleaned from the patient's record or discharge summary at the Hospital (HRAS).

Data analysis

For comparison of multiple variables, the Student's t test was used for independent samples, Fisher's exact test and Chi Square were also applied. The null hypothesis of equality of the measurements and a significance level of 5% were used.

RESULTS

The distribution of children according to age at the time of clinical examination, gestational age, birth weight and gender are shown in Table 1.

Total numbers of teeth present (incisors, canines and molars) at the time of examination and the distribution by groups is described in Table 2.

Hypoplasia was more frequent in the preterm group. Opacities, however, were more frequent in the full term group (Table 3). The distribution of enamel defects by groups is also shown in Table 3.

Opacity occurred in 28.1% of G1 children and 18.8% of G2 children (Table 4). The difference between these percentages was not statistically significant ($p = 0.173$). Hypoplasia was found in 8.3% and 37.5% of G1 and G2 children, respectively. This difference was statistically significant ($p \leq 0.001$) (Table 4).

There was no need for ventilatory assistance in 100% of the full term group. Data on preterm children who required ventilatory assistance and the presence of enamel defects are shown in Table 5.

Parenteral nutrition (PN) was also present only in premature children. A total of 12.3% of children that received PN had opacities, compared with 87.7% that also received PN but did not have opacities (p=0.64). Further, 56.1% of children with PN were diagnosed with hypoplasia (p=0.001) (Table 6).

The preterm group was categorized as VLBW (very low birth weight, up to 1500g), LBW (low birth weight, between 1500g and 2500g) and NBW (normal birth weight, greater than 2500g). Data pertaining to the defects that were found are shown in Table 7.

Table 1. Mean number of full term and preterm children in terms of age, gestational age, birth weight and distribution according to gender.

		Full Term		Preterm		P
		Mean	Standard Deviation	Mean	Standard Deviation	
Age (months)		40.72	16.33	30.44	14.43	<0.001
Gestation period-weeks		38.93	1.17	30.63	2.90	<0.001
Birth weight (grams)		3294.3	501.06	1421.3	601.91	<0.001
		n	%	N	%	
Gender	Fem	45	46.9	54	56.3	0.124
	Male	51	53.1	42	43.8	

Data on mean defective teeth per child and categorization of these infants by weight are presented in Table 8.

DISCUSSION

The results showed that children born prematurely (G2) were hospitalized for a period ranging from 0 to 120 days. The most frequent neonatal complications were: respiratory distress, jaundice, pneumonia, osteopenia of prematurity, anemia and nonspecific infections. Several drug therapies, such as antibiotics, also ventilatory support (mechanical ventilation, continuous positive airway pressure (CPAP)), parenteral nutrition and prescription of vitamin supplements containing iron and calcium were required. The need for these procedures was directly related to the state of the each child's general health.

No significant differences in the prevalence of mineralization disorders were found when children from G1 and G2 were divided by gender. The values were, respectively, P = 0.115 and P = 1.000 for opacity and hypoplasia. The findings corroborate those of Mellander *et al* (1992)[13], Kanchanakamol *et al* (1996)[14] and Lima *et al* (1999)[15], who also found no differences.

The average age of the children was 40.72 months for the full term group, and 30.44 months for the premature infants. The gestational age was 38.93 weeks for the full term group and 30.63 for the premature group. Birth weight was 3294 grams and 1421 grams for the full term and preterm groups, respectively. A total of 1710 teeth were examined in the full term group and 1388 teeth in the premature group. This difference may be explained by the fact that in the preterm group there was a delay in the eruption of teeth.

Children born prematurely showed more enamel defects compared with children born at full term. Sixty-four defective teeth were found in G1 (51 opacities and 13 hypoplasias), and 110 in G2 (29 opacities and 81 hypoplasias). The results were significant only for hypoplasia (Table 3), and corroborate the work of Aine *et al*[5] and others)[8,16,17] who also found significant results only for hypoplasia. Other types of defects (agenesis, supernumerary and twinning) were found only in G1 (Table 3).

In the total sample, the groups of teeth most affected by hypoplasia were incisors (70.2%), canines (21.3%) and molars (8.5%). Similar findings were reported by Li *et al*.[18] The lower prevalence of hypomineralization and hypoplasia in molars is due to the period of mineralization that occurs following the period of intubation, which usually happens early in life.[18] Concerning opacity, there was a reversal of prevalence in the sense that the groups with the most affected teeth were canines (48.8%), molars (30%) and incisors

Table 2. Number of teeth present and distribution by group

	No. Teeth	Upper Incisors	Lower Incisors	Upper Canines	Lower Canines	Upper Molars	Lower Molars
Full Term	1710	377	371	170	168	306	318
Preterm	1388	355	343	125	122	218	225

Table 3. Number and percentage of teeth with enamel defects in full term and preterm groups.

		Full Term (G1)		Preterm (G2)	
		Number	Percentage	Number	Percentage
Opacity	Incisor	4	6.2	13	11.8
	Molar	17	26.6	7	6.4
	Canine	30	46.9	9	8.2
Hypoplasia	Incisor	4	6.2	62	56.4
	Molar	3	4.7	5	4.5
	Canine	6	9.4	14	12.7
Total		64	100	110	100

Table 4. Percentage of children in the full term and preterm groups who presented with opacity and hypoplasia.

		Full Term (G1)		Preterm (G2)		p
		n	%	n	%	
Opacity	Yes	27	28.1	18	18.8	0.173
	No	69	71.9	78	81.3	
Hypoplasia	Yes	8	8.3	36	37.5	<0.001*
	No	88	91.7	60	62.5	

Table 5. Relationship between hypoplasia and opacity and the need for ventilatory support in preterm children.

		Ventilatory Assistance				P
		No		Yes		
		N	%	N	%	
Opacity	No	12	80.0	66	81.5	1.000
	Yes	3	20.0	15	18.5	
Hypoplasia	No	15	100.0	45	55.6	0.001*
	Yes	0	0	36	44.4	

(21.3%) (Figure1). The groups of teeth most affected by tooth enamel defects were canines (full term group) and incisors (premature group). The higher prevalence of defects in incisors and canines can be explained by the chronology of tooth mineralization, which occurs approximately around the 9th month of gestation.

A statistically significant higher number of children from the premature group showed hypoplasia when compared to the full term group (P≤ 0.001 table 4). This data may again point to prematurity as an etiological factor for these defects, and is in accordance with Klinberg *et al* and Seow *et al* (1990).

The prevalence of enamel defects (hypoplasia and opacity) was analyzed in preterm and full term groups. Children were classified according to their birth weight as very low, low and normal birth weight. In the preterm and very low birth weight group (VLBW < 1500g), 51.4% presented hypoplasia. This result reflects a statistically significant difference in prevalence ($p < 0.001$): i.e., weight may be a factor related to increased prevalence of defects in these children (Table 6). In this study, only hypoplasia correlated positively with very low birth weight. Seow *et al* [20] and others[15,17] also found that preterm children with low birth weight (LBW <2500g) also exhibited a higher prevalence of defects than children with normal weight (NBW > 2500g). Children born with very low weight usually experience more neonatal complications than children born with low or normal weight. These intercurrent events can be listed as probable causes of enamel defects. The most common complications were: nonspecific infections, respiratory diseases, mineral deficiencies, bone disease of prematurity, among others.[21-23] Calcium absorption by the human embryo is at its peak in the last trimester of pregnancy.[16] Since these children were born before this period was completed, they are likely to have suffered from calcium deficiency. As a result, tooth enamel, as well as other body tissues, may have been adversely affected.

A significant relationship was found among the group of children who required ventilatory support and parenteral nutrition in the presence of hypoplasia. One hundred percent of the 36 children who had hypoplasia needed ventilatory assistance (VA); and, 34 of them needed parenteral nutrition (PN). Opacity did not display a significant relationship (Tables 5 and 6). Although a strong association between these factors was found, some of the children who had enamel defects did not require VA and NP. It can be inferred that other factors might also play a role in the etiology of these defects.

In most cases, the endotracheal tubes used for ventilation are maneuvered and positioned in the region of the maxilla. The force exerted by the pressure of these tubes can be excessive, and can cause enamel defects and deformities of the palate.[24] They may even damage dental crowns. In G2, the percentages of enamel defects

Table 6. Relationship between parenteral nutrition and the presence of opacity and hypoplasia in the premature group.

		Nutrition				Total		
		N		S				
		n	%	N	%	n	%	
Opacity	No	28	71.8	50	87.7	78	81.3	0.64
	Yes	11	28.2	7	12.3	18	18.8	
Hypoplasia	No	35	89.7	25	43.9	60	62.5	0.001*
	Yes	4	10.3	32	56.1	36	37.5	

Table 7. Relationship between the presence of enamel defects in the preterm group and birth weight.

		Categorized Birth weight								p
		Very low		Low		Normal		Total		
		n	%	n	%	n	%	N		
Opacity	Yes	13	18.6	3	15.8	2	28.6	18	18.8	0.758
	No	57	81.4	16	84.2	5	71.4	78	81.3	
Total		70	100.0	19	100.0	7	100.0	96	100.0	
Hypoplasia	Yes	36	51.4	0	0	0	0	36	37.5	<0.001*
	No	34	48.6	19	100.0	7	100.0	60	62.5	
Total		70	100.0	19	100.0	7	100.0	96	100.0	

Table 8. Proportion of teeth with enamel defects per child between the full term and preterm groups, and by birth weight in the premature group.

	Number of teeth with	Group	N	Mean	Standard Deviation	p
	Opacity	Full Term	96	0.0277	0.05301	0.645
		Preterm	96	0.0321	0.07770	
	Hypoplasia	Full Term	96	0.0073	0.02618	<0.001
		Preterm	96	0.0599	0.10628	
Preterm	Opacity	Very low	70	0.0346	0.07995	0.449
		Low	19	0.0147	0.04088	
		Normal	7	0.0548	0.12424	
	Hypoplasia	Very low	70	0.0822	0.11703	0.003
		Low	19	0.0000	0.00000	
		Normal	7	0.0000	0.00000	

were nearly equal in the maxilla and in the mandible (49% and 51% for opacity, and 46.2% and 53.8% for hypoplasia, respectively). Also in G2, we found a predominance of defects in the maxilla (Figure 2). It is likely that this greater prevalence of defects is linked to the ventilatory maneuvers. The results found in this study corroborate the work of Seow,[9] and others [5,10,18] who found a predominance of defects in the maxilla and attributed them to traumatic laryngoscopy during primary teeth mineralization.

It is difficult to determine a specific cause for the occurrence of enamel defects in children born prematurely. Since several factors operate concurrently, the scenario is too complex, precluding the suggestion of any specific order of importance. In attempting to pinpoint the probable etiologic factors of these defects, a detailed history of the gestational period is of paramount importance, including data on birth and neonatal complications.

CONCLUSIONS

Within the limitations of this case-controlled study, it was concluded that:

1- Children born prematurely show a higher prevalence of enamel defects, compared to children born at full term.

2- Hypoplasia was the more prevalent defect.

3- Frequency of enamel defects was found to be higher among very low birth weight (VLBW) children, compared to low birth weight (LBW) and normal birth weight (NBW) children. Ventilatory support and parenteral nutrition were two other risk factors we were able to correlate with the presence of enamel defects.

REFERENCES

1. Rades E, Bittar RE, Zugaib M - Direct Determinants of Elective Preterm Birth and Neonatal Results. *Rev Bras Ginecol Obstet.* (online) *26*(8):665-62. 2004.
2. Ferrini FRDO, Marba STM, Gavião MBD. Buccal problems in premature and low birth weight children. *Rev. Paul. Pediatr; 25*(1): 66-71. 2007.
3. Gravina DBL, Cruvinel VRN, Azevedo TDPL, Bezerra ACB, Toledo OA. Prevalence of dental caries in children born prematurely or at full term. *Braz Oral Res; 20*(4) :353-7. 2006.
4. FDI Commission on Oral Health- World Dental Press. A review of the developmental defects of enamel index (DDE index). *Int Dent J; 42* (6): 411-428. 1992.
5. Aine L. *et al*. Enamel defects in primary and permanent teeth of children born prematurely. *J Oral Pathol Med; 29*(8): 403-9. 2000.
6. Lai PY, Seow WK, Tudehope DI, Rogers Y. Enamel hypoplasia and dental caries in very-low birthweight children: a case-controlled, longitudinal study. *Pediatr Dent; 19*(1): 42-9. 1997.
7. Moylan FMB, Seldin EB, Shannon DC, Todres ID. Defective primary dentition in survivors of neonatal mechanical ventilation. *J Pediatr; 96*(1): 106-8. 1980.
8. Fearne JM *et al*. Enamel defects in the primary dentition of children born weighing less than 2000g. *Br Dent J; 168* (11): 433-437. 1990.
9. Seow WK, Perham S. Enamel hypoplasia in prematurely-born children: A scanning electron microscopic study. *J Pedod; 14*(4):235-9. 1990.
10. Angelos GM, Smith DR, Jorgenson R, Sweeney EA. Oral complications associated with neonatal oral tracheal intubation: a critical review. *Pediatr Dent; 11*(2):133-140. 1989.
11. Seow WK, Brown JP, Tudehope DI, O Calaghan M. Developmental defects in the primary dentition of low birth-weight infants: adverse effects of laryngoscopy and prolonged endotracheal intubation. *Pediatr Dent; 6*(1):28-31. 1984.
12. Horowitz HS. Research issues in early childhood caries. Community *Dental Oral Epidemiol; 26*(1 Suppl): 67-81.1988.
13. Mellander M, Noren JG, Fredén H, Kjellmer I. Mineralization defects in deciduous teeth of low birthweight infants. *Acta Paediatr Scand; 71*(5): 727-33. 1982.
14. Kanchanakamol U *et al*. Prevalence of developmental enamel defects and dental caries in rural pre-school Thai children. *Community Dental Health; 13*(4):204-207. 1996.
15. Lima MGGC, Duarte RC – Prevalence of the enamel defects in children with low weight at birth, in the group from 6 to 72 months in Joao Pessoa city. J Bras. Odontopediatr . *Odontol Bebe; 2*(10): 459-467. 1999.
16. Drummond BK, Ryan S, O`Sullivan EA, Congdon P, Curzon MEJ. - Enamel defects of the primary dentition and osteopenia of prematurity. *Pediatr Dent; 14*(2):119-21. 1992.
17. Li Y, Navia JM, Bian JY. Prevalence and distribution of developmental enamel defects in primary dentition of Chinese children 3-5 years old. *Community Dent Oral Epidemiol; 23*(2): 72-9. 1995.
18. Noren JG, Ranggard L, Klinberg G, Persson C, Nilsson K. Intubation and mineralization in the enamel of primary teeth. *Acta Odontol Scand; 51*(5): 271-5. 1993.
19. Klinberg G, Oskarsdottir S, Johannesson EL, Noren JG. Oral manifestations in 22q11 deletion syndrome. *Int J Paediatr Dent; 12* (1): 14-23. 2002.
20. Seow WK *et al*. Enamel hypoplasia in the primary dentition: a review. *ASDC J Dent Child ; 58*(6):441-52. 1991.
21. Pimlott JFL, Howey TP, Nikiforuk G, Fitzhardinge PM. Enamel defects in prematurely born, low birth-weight infants. *Pediatr Dent; 7*(3):218-23. 1985.
22. Seow WK, Humphrys C, Tudehope DI. Increased prevalence of developmental dental defects in low-birth-weight, prematurely born children: a controlled study. *Pediatr Dent; 9*(3): 221-225. 1987.
23. Gerlach RF; Sousa MLR; Cury JA. Defective Enamel: from biological marker to clinical implications – Rev Odonto Ciênc; 15(31): 87-102. 2000.
24. Seow WK. Effects of preterm birth on oral growth and development. *Austr Dent J; 42*(2): 85-91. 1997.