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Dental Findings in Primary Dentition of Very Preterm Children: A Retrospective Case-Control Study from Finland

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Abstract

Objective: The aim of this study was to compare the prevalence of dental deviations in primary dentition in very preterm and full-term children.

Material and methods: The subjects consisted of 205 very preterm and 205 full-term children. Data were combined from two sources: (1) the register from the Turku University Hospital of children born before the 32nd week of pregnancy or with a birth weight of less than 1,500 g, and (2) public health centre dental registers. All included very preterm (case) children were born in the Turku University Hospital during 2001-2006, and the full-term control children were matched according to age and gender.

Results: The prevalence of enamel defects of the all primary teeth was 2.10% in case children and 0.42% in control children (RR= 5.07, 95% CI 1.96-13.13, p=0.001). The prevalences of having at least one enamel defect were 10.73% in case children and 4.39% in control children (RR=2.44, 95% CI 1.22-4.91, p=0.012). The prevalences of missing teeth, supernumerary teeth and teeth with shape anomalies were low in both groups.

Conclusions: The results confirm earlier findings that preterm children have a higher incidence of enamel defects in primary dentition compared to full-term children. More studies are needed to elucidate the effect of preterm birth on the number and shape of developing teeth.

Keywords: Dental anomaly; Enamel defect; Missing tooth; Premature birth; Primary dentition; Register study; Supernumerary tooth; Tooth anomaly

Introduction

According to the World Health Organization, children who are born before 37 weeks of pregnancy or weigh less than 2,500 g are born preterm. Extremely preterm children are born before the 28th week of pregnancy, very preterm between the 28th and 32nd weeks of pregnancy, and moderate to late preterm between the 32nd and 37th weeks of pregnancy. The most common causes for premature birth are multiple pregnancies, infections and chronic conditions, e.g., diabetes. Often, however, no specific reason can be identified. Every year, roughly 15 million children worldwide are born prematurely, most of them in developing countries [1]. In Finland, about 5-6% of all births are premature; approximately 1% of children are born very preterm [2]. Today, the survival rate of preterm children in Finland is high, and any dentists working at a public health centre encounter such children in their practice.

Premature birth affects many organs, and serious complications can also occur in the mouth [3]. The most common developmental disturbances in dentition are enamel defects. They can be divided into hypoplasia and opacity. Hypoplasia manifests as pits, grooves or areas of missing enamel, while opacity appears as white, creamy or yellow-to-brownish colouring [4]. Most hypoplasias are situated in the maxilla [5]. According to a systematic review, three out of four studies reported an association between prematurity and enamel hypoplasia in primary dentition [6]. In preterm children, the prevalence has been found to vary from 43 % to up to 96 % [5,7,8]. Hypoplasia is more common in primary than in permanent dentition, because primary dentition is undergoing mineralization around the time of premature birth [9].

There are no published studies evaluating dental anomalies such as hypodontia (congenital lack of one or more teeth), hyperdontia (presence of one or more supernumerary teeth) or abnormal tooth shape (usually double or peg-shaped teeth) in preterm children. Because nutritional deficiencies, exposure to medications, or oral manipulations can affect oral development in preterm children, it is plausible that these factors may also predispose to dental deviations, e.g. abnormally shaped or missing teeth. Previous studies on dental anomalies in full-term children have shown that malformed teeth are less common in primary than in permanent dentition, except for double teeth, which are likely more common in primary than in permanent dentition [10,11]. The aim of this study was to compare

dental findings in primary dentition between very preterm and full-term children. The hypothesis was that very preterm birth has significant consequences for developing dentition.

Material and Methods

The present case-control study is part of the Finnish multidisciplinary PIPARI Study (Development and Functioning of Very Low Birth Weight Infants from Infancy to School Age, Turku University Hospital, Finland). Data were retrospectively collected from two sources: (1) the register from the Turku University Hospital of children born preterm (before the 37th week of pregnancy) with a birth weight of less than 1,500 g between 2001 and 2003, and between early 2004 and 2006, when the inclusion criteria were broadened to include all infants born before the 32nd week of pregnancy, regardless of birthweight; and (2) the dental registers of children in public dental care. In Finland, dental care is provided free of charge to all children and adolescents aged 0-18 years, and dental examinations take place on a regular basis. Children with no obvious risk factors are called in individually either annually or every second-to-third year for dental examinations. Children needing follow-ups e.g., for dental trauma or for occlusal development and children with high risk for caries are called in up to two to three times per year, depending on the individual risk factors. The examination data is stored in electronic dental records as dental status and supplementary written text.

During 2001-2006, a total of 290 children who met our inclusion criteria were born very preterm in the Turku University Hospital. Age- and gender-matched control children were found in the register of the City of Turku Oral Health Care (Health and Social Services). Every case child had 3-4 potential age- and gender-matched full-term control children (n=636). The first control child in alphabetical order was included as a control child (n=205). If the control child was born preterm, was diagnosed with some syndrome or craniofacial anomaly, or had no dental information, the next child in order was included.

The final case and control groups consisted of 205 very preterm (Figure 1) and 205 full-term control children, with 90 girls and 115 boys in each group. Maternal history was not obtained. When intubation had been used during the children's perinatal hospital care, it had always been done through the nose.

One of the examiners gathered and coded all the data from the electronic dental records. The examination data were stored in electronic dental records as dental status and supplemented with written text (e.g. description of the hypoplasia). Primary dentition data included information on gender, enamel defects (per tooth and surface), abnormal tooth shape, and hypo- and hyper-dontia. Data were collected from every dental examination before the beginning of the early mixed dentition stage. If a finding was present, e.g. in the first examination but not in the second, and then reappeared in the next examination, the finding was included in the data. If a finding was present in the first examination but did not reappear in later examinations, the finding was not included in the data. In the majority of cases, where radiographs were needed, orthopantomographs had been taken, e.g. to assess the number of supernumerary and/or missing teeth.

The PIPARI Study protocol was approved by the Ethics Committee of the Hospital District of Southwest Finland in December 2000 and January 2012.

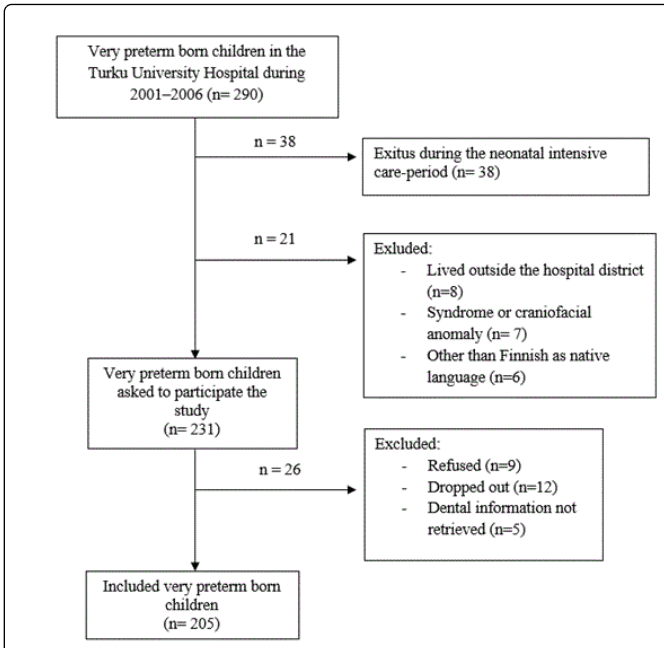

Figure 1: A flow-chart of collecting the case children.

Statistical methods

The prevalences of enamel defects of the all the primary teeth in case and control groups were compared with negative binomial regression using generalized estimating equations to account for the case-control matched study design [12]. The prevalence of having at least one enamel defect was also calculated. Results are expressed as relative risks (RR) with 95% confidence intervals (CI). Statistical analyses were performed using IBM SPSS Statistics for Windows, Version 25 (IBM Corp., Armonk, NY). P-values of less than 0.05 were interpreted as statistically significant.

Results

All the observed dental deviations among the case group and the control group according to location of deviation (maxilla and mandible) are shown in Table 1.

Enamel defects

Among the 22 case children the defects were registered in 86 primary teeth. In the control group, defects affected a total of 17 teeth in 9 children. Total number of teeth was 4088 in control group and 4092 in case group. In case children, the prevalence of enamel defects of all the primary teeth was 2.10% and in control children 0.42% (RR=5.07, 95% CI 1.96-13.13, p=0.001).

10.73% (22/205) of the children in case group and 4.39% (9/205) of the children in control group had at least one enamel defect (RR=2.44, 95% CI 1.22-4.91, p=0.012).

In the case group, most of the defects were in the maxilla (Table 1). Most of the case children had one or two enamel defects in the maxilla and/or one defect in the mandible, whereas most of the control children only had one enamel defect in either or both jaws.

Case girls had more enamel defects than case boys (57/1794 vs. 29/2298 defects/total number of primary teeth, respectively) but the difference in the prevalence of enamel defects of all the primary teeth between girls and boys was not statistically significant (3.18% vs. 1.26%, RR=2.53, 95% CI 0.80-8.04, p=0.114). Of the 57 enamel defects in case girls, 40 were registered in two girls with a defect in every primary tooth. 10.0% (9/90) of the girls in case group and 11.3% (13/115) of the boys in case group had at least one enamel defect (RR=0.89, 95% CI 0.40-1.98, p=0.765).

In the control group, the difference in the prevalence of enamel defects between girls and boys (8/1793 vs. 9/2295 defects/total number of primary teeth, respectively) was negligible (0.45% vs. 0.39%, RR=1.14, 95%, CI 0.28-4.57, p=0.855). 4.44% (4/90) of the girls in control group and 4.35% (5/115) of the boys in control group had at least one enamel defect (RR=1.02, 95% CI 0.28-3.70, p=0.973).

Case Group			
Dental deviation	Maxilla	Mandible	Total
Enamel defects	56	30	86
Malformed teeth	6	3	9
Missing teeth	1	-	1
Control Group			
Dental deviation	Maxilla	Mandible	Total
Enamel defects	9	8	17
Malformed teeth	3	3	6
Missing teeth	-	2	2

Table 1: The number of affected primary teeth in case children (n=205) and control children (n=205).

The distributions of the defects in case and control groups are presented in Table 2 and the distributions according to gender are presented in Table 3.

Case children									
Upper right									Upper left
5 d55	2 d54	4 d53	5 d52	7 d51	10* d61	9 d62	4 d63	3 d64	7 d65
d85 3	d84 3	d83 3	d82 2	d81 3	d71 4	d72 4	d73 2	d74 2	d75 4
Lower right									Lower left
Control children									
Upper right									Upper left
1 d55	- d54	2 d53	- d52	- d51	- d61	1 d62	2 d63	- d64	3* d65
d85 1	d84 2	d83 -	d82 -	d81 -	d71 -	d72 -	d73 2	d74 1	d75 2
Lower right									Lower left
*The most frequently affected teeth									

Table 2: The number of enamel defects per tooth in (a) Case children (n=205) and (b) Control children (n=205) presented in dental status form. The most frequently affected teeth marked with symbol *.

Case girls									
Upper right									Upper left
4 d55	2 d54	2 d53	3 d52	4 d51	4 d61	3 d62	2 d63	2 d64	5* d65
d85 2	d84 2	d83 3	d82 2	d81 3	d71 4	d72 3	d73 2	d74 2	d75 3
Lower right									Lower left
Case boys									
Upper right									Upper left
1 d55	- d54	2 d53	2 d52	3 d51	6* d61	6* d62	2 d63	1 d64	2 d65
d85 1	d84 1	d83 -	d82 -	d81 -	d71 -	d72 1	d73 -	d74 -	d75 1
Lower right									Lower left
Control girls									
Upper right									Upper left
- d55	- d54	1 d53	- d52	- d51	- d61	1 d62	1 d63	- d64	1 d65
d85 1	d84 1	d83 -	d82 -	d81 -	d71 -	d72 -	d73 1	d74 -	d75 1
Lower right									Lower left
Control boys									
Upper right									Upper left
1 d55	- d54	1 d53	- d52	- d51	- d61	- d62	1 d63	- d64	2* d65
d85 -	d84 1	d83 -	d82 -	d81 -	d71 -	d72 -	d73 -	d74 1	d75 1
Lower right									Lower left
*Most frequently affected teeth									

Table 3: The number of enamel defects per tooth in (a) Case girls (n=90), (b) Case boys (n=115), (c) Control girls (n=90) and (d) Control boys (n=115) presented in dental status form. The most frequently affected teeth marked with symbol *.

Malformed teeth and missing teeth

Case children had nine and control children six malformed primary teeth of all teeth (Table 1). In case children, the prevalence of malformed teeth of the all primary teeth was 0.22% and in control children 0.15% (RR=1.50, 95% CI 0.30-7.48, p=0.622). In most cases, there were only one or two malformed teeth per child, with the exception of one case child with four malformed teeth in the maxilla (from maxillary left central incisor to maxillary left first molar; d61-d64). In addition, case children had three and control children four double teeth. One control child had two double teeth (mandibular left canine and mandibular right lateral incisor; d73, d82). In the control group, two teeth were described as peg-shaped (d52, d54).

In one case child, the maxillary left canine (d63) was missing, while in two control children, two teeth were missing (one mandibular right central incisor and one mandibular right canine; d81, d83) (Table 1).

In case children, a total of four supernumerary teeth were found; three of them were located between maxillary central incisors (i.e., so-called mesiodentes). In control children, the number of supernumerary teeth was two.

Discussion

Nearly 60 papers from the Finnish PIPARI study have been published, but no dental studies as of yet. The aim of this study was to

compare dental findings in primary dentition in very preterm and full-term children. So far, no previous studies have focused on dental anomalies such as hypodontia, hyperdontia or malformed primary teeth in prematurely born children.

In a recent review, three out of four studies found an association between enamel hypoplasia and preterm birth; in two out of four studies, opacity was significantly more common in preterm than in full-term children [6]. The results of the present study confirm earlier findings: the prevalence of enamel defects among very preterm children was fivefold compared to that among full-term children.

Enamel hypoplasia can be generalized or localized. In the generalized form, the defects are symmetrical and most likely caused by systemic factors [8], while in the localized form, some local factor, e.g., trauma, has caused the hypoplasia. In previous studies, oral intubation has been suggested as a cause of local trauma, leading to disturbances in upper incisors [5,9]. Thus, oral intubation has been commonly replaced by nasal intubation [6]. However, although none of the case children in this study had undergone oral intubation, the anterior part of the upper jaw was still the most affected. Presumably, this is due to the temporal relation between birth and stage of tooth development. Given that the mineralization of primary incisor crowns is nearly completed near the 40th week of gestation [8], all of the case children had already been born weeks before this time point. However, it is also possible that systemic factors played a role in the development of enamel defects. For example, respiratory distress syndrome, hypoxia and hyperbilirubinemia have been reported to increase the prevalence of enamel defects in primary dentition by interfering with calcium homeostasis [5].

Enamel is the only hard tissue in the human body that does not remodel after development [4]. Once an injury occurs on the tooth's surface, its imprint becomes permanent [7,8]. Seow and team [13] found that prematurely born children have approximately 20% thinner enamel than their full-term controls, and this deficiency was observed in the prenatally formed enamel. The so-called "catch-up" enamel that forms after birth does not fully compensate for the deficiency in prenatal enamel. The main problems related to enamel hypoplasia include dental caries and plaque accumulation, and in visible areas, possible aesthetic issues [8]. Because enamel is not as fully developed as it should be, dental caries can proceed faster in hypoplastic than in normal teeth. At worst, it can lead to extraction of teeth and thereby to loss of space in primary dentition [8].

Although double teeth are more common in primary than in permanent dentition [11], they were rare findings in this study: there were only three references to double teeth in case children and four in control children. By contrast, hyperdontia has been found to be less common in primary than in permanent dentition [11]. It is usually diagnosed by accident during intraoral examinations or in radiographs, and can cause, e.g. malocclusion, eruption disturbances, crowding or retention of teeth [14]. In the present case group, three of the four supernumerary teeth were so-called mesiodentes. Occasionally, they are also found in permanent dentition, although they cannot be distinguished specifically as either "primary" or "permanent" teeth. The share of missing primary teeth was low in both case and control groups when compared to the corresponding prevalence in Finnish full-term children (0.9%) [15].

In the present study, all of the 205 case children were born in the same hospital and treated according to the same principles during their perinatal hospital care, which can be considered a strength of this study. However, there were also limitations. First, data were retrospectively collected from dental registers and covered individual time periods of approximately three years (corresponding with children's primary dentition stage). All markings were based on children's dental examinations made by clinicians during their normal, everyday work; no special study criteria or calibrated measures were available. The data were stored in electronic dental records as dental status and supplementary written text, describing e.g., the features, size and position of hypoplasia. Although it is probable that there was some variation in documentation, similar limitation applies to both case and control children. Secondly, inclusion of more control children would have improved the statistical power of our findings. However, when designing this study, one control child per one case child was considered adequate, because the sample size well exceeded one hundred. Nevertheless, the results should be interpreted with caution.

Conclusion

Our results confirm earlier findings that prematurely born children have a higher incidence of enamel defects in primary dentition compared to full-term children. These defects most frequently affect upper incisors, which may be a consequence of temporary coincidence between birth and tooth development. More studies are needed to elucidate the prevalence of other dental anomalies in preterm children.

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Conflict of Interest

Petra Lüthje, Tero Vahlberg, Elina Maaniitty, Päivi Rautava and Anna-Liisa Svedström-Oristo hereby state that they have no conflicts of interest.

References

1. World Health Organization (2017) Preterm birth. WHO, Geneva, US.
2. Current Care Guidelines (2018) Preterm birth. Current Care Summary. The Finnish Medical Society Duodecim, Helsinki, Finland.
3. Seow WK (1997) Effects of preterm birth on oral growth and development. Aust Dent J 42: 85-91.
4. ZaidiI I, Thayath MN, Sinhg S, Sinha A (2015) Preterm birth: a primary etiological factor for delayed oral growth and development. Int J Clin Pediatr Dent 8: 215-219.
5. Aine L, Backström MC, Kuusela AL, Koivisto AM, Ikonen RS, et al. (2000) Enamel defects in primary and permanent teeth of children born prematurely. J Oral Pathol Med 29: 403-409.
6. Jacobsen PE, Haubek D, Henriksen TB, Østergaard JR, Poulsen S (2014) Developmental enamel defects in children born preterm: a systematic review. Eur J Oral Sci 122: 7-14.
7. Cruvinel VRN, Gravina DBL, Azevedo TDPL, De Rexende CS, Bezerra ACB, et al. (2012) Prevalence of enamel defects and associated risk factors in both dentitions in preterm and full term born children. J Appl Oral Sci 20: 310-317.
8. Eastman DL (2003) Dental outcomes of preterm infants. Newborn Infant Nurs Rev 3: 93-98.
9. Paulsson L, Bondemark L, Söderfelt B (2004) A systematic review of the consequences of premature birth on palatal morphology, dental occlusion, tooth-crown-dimensions and tooth maturity and eruption. Angle Orthod 74: 269-279.

10. Marinelli A, Giuntini V, Franchi L, Tollaro I, Baccetti T, et al. (2012) Dental anomalies in the primary dentition and their repetition in the permanent dentition: a diagnostic study. Odontology 100: 22-27.
11. Mukhopadhyay S, Mitra S (2014) Anomalies in primary dentition: Their distribution and correlation with permanent dentition. J Nat Sci Biol Med 5: 139-143.
12. Hardin JW, Hilbe JM (2012) Generalized Estimating Equations. (2ndedn), Chapman & Hall/CRC, UK.
13. Seow WK, Young WG, Tsang AK, Daley T (2005) A study of primary dental enamel from preterm and full-term children using light and scanning electron microscopy. Pediatr Dent 27: 374-379.
14. Chaudhary S, Chaitra TR, Sultan S, Arora R (2013) Supernumerary teeth in primary dentition. BMJ Case Rep 2013: bcr2013200029.
15. Järvinen S, Lehtinen L (1981) Supernumerary and congenitally missing primary teeth in Finnish children. An epidemiologic study. Acta Odontol Scand 39: 83-86.