Enamel of Premolars whose Predecessors Presented Rupture of the Follicle Bone Crypt from Periapical Infections

Marcondes Cavalcante Santana Neto*/ Ana Victória Lopes Bandeira**/ Heloísa Clara Santos Sousa***/ Marcoeli Silva de Moura****/ Lúcia de Fátima Almeida de Deus Moura*****/ Marina de Deus Moura de Lima ******

Objective: *To observe if dental alterations in premolars are associated with the rupture of the follicle bone crypt caused by a periapical lesion in the predecessor molars.* ***Study design:*** *This is a cross-sectional study. Data collection consisted of the analysis of medical records, a socioeconomic questionnaire, and dental clinical examination. Records from patients with a history of a radiographically visible periapical lesion in lower primary molars submitted to pulp therapy or extraction from a university dental clinic were selected. Successor premolars were clinically evaluated for the presence of enamel development defects, shape alterations, and eruption deviations. The descriptive analysis of data was performed, and Fisher's exact tests, linear trend chi-square, and the Student's t-test were applied.* ***Results:*** *Forty-eight permanent teeth were from 36 patients were evaluated in this study, and 20 (41.7%) of the 48 examined premolars showed a radiographic image suggestive of the rupture of the bone crypt. Rupture of the follicle bone crypt was not associated with the occurrence of enamel alterations in premolars (p = 0.418). An association between dental alterations and age over six years at the time of intervention was observed (p = 0.043).* ***Conclusion:*** *The presence of enamel alterations of premolars was not associated with the rupture of the follicle bone crypt caused by a periapical lesion in predecessor molars.*

Keywords: *Pulpectomy, Primary Tooth, Tooth Abnormalities, Permanent Dentition.*

INTRODUCTION

Primary teeth and their successors form distinct but interdependent units.[1] Traumatic injuries or pulp infections may interfere with the homeostasis of these units due to the close anatomical proximity between them and the metabolic alterations through pulpoperiodontal communications.[1-3] Thus, lesions involving periradicular tissues of the primary teeth may be a factor associated with alterations in dental developing enamel.[1,2,4-6]

The pulp chamber floor of primary molars presents particularities, such as reduced dentin thickness, areas with different stages of resorption and the presence of accessory canals.[7] These conditions predispose the communication of the coronary pulp with the periodontal region, where the successor tooth is in development.[8,9] These communication pathways may justify the presence of radiolucent areas in inter-root regions of primary molars with pulp infections.[10]

Pulpal therapy is the procedure of choice for preserving primary teeth with irreversible pulpitis and pulp necrosis[11-13], as it favors the maintenance and restoration of masticatory function, the integrity of periradicular tissues and the preservation of the space for permanent successor eruption.[11,12,14] But in cases of predecessor teeth with chronic periapical lesions that have caused the rupture of the bone crypt of the permanent successor in formation, extraction is indicated.[1,2,12,13] However, there is no scientific evidence to justify

From the Department of Pathology, School of Dentistry, Universidade Federal do Piauí–UFPI, Teresina, Brazil.
* Marcondes Cavalcante Santana Neto, DDS.
**Ana Victória Lopes Bandeira, DDS, MsC .
***Heloísa Clara Santos Sousa, DDS, MsC.
****Marcoeli Silva de Moura, DDS, MsC, PhD, Full time Professor.
*****Lúcia de Fátima Almeida de Deus Moura, DDS, MsC, PhD , Full time Professor.
******Marina de Deus Moura de Lima, DDS, MSc, PhD, Adjunct Professor.

Send all correspondence to:
Marina de Deus Moura de Lima
Department of Pathology and Dentistry Clinic, School of Dentistry, Universidade Federal do Piauí Campus
Ministro Petrônio Portella–Block 5 Ininga District, 64049-550. Teresina–Piauí–Brazil. Phone: +55 86 3232 5928 / +55 86 99964 1710
E- mail: mdmlima@gmail.com

this procedure. In addition, the early loss of primary molars can trigger anatomic (hypoplasia and hypocalcification), masticatory, and orthodontic problems (eruption disorders and loss of space in the dental arch).[14]

Enamel development and the eruption of premolars, when the rupture of the predecessor molar bone crypt occurs by periapical injury have not been studied. The knowledge of the possible effects will serve as a scientific basis for adopting appropriate treatment, considering the possible associated sequelae. The aim of this study was to observe if dental alterations in the premolars are associated with the rupture of the follicle bone crypt caused by a periapical lesion in the predecessor molars.

MATERIALS AND METHOD

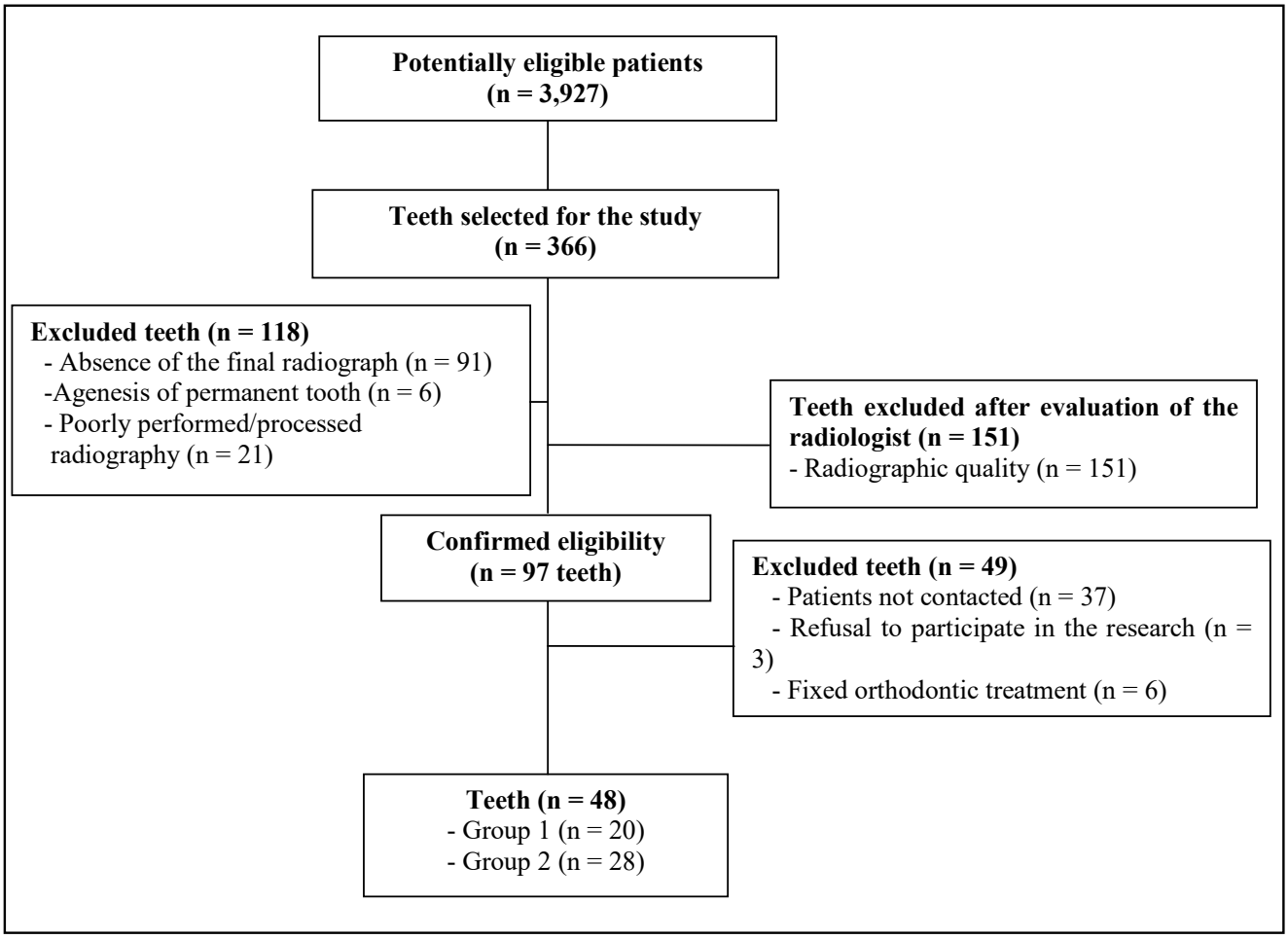
This cross-sectional study was approved by the Research Ethics Committee (REC) of the Federal University of Piauí (UFPI) (Protocol No. 1035345) and was conducted in accordance with the Declaration of Helsinki. Written consent was obtained from the study participants and their caregivers.

The sample was composed of children and adolescents attending the UFPI children's dental clinic, selected for review of all the medical records (n = 3,927). Children aged over eight years old, who presented a history of lower primary molars with a periapical lesion visible on the radiograph, submitted to pulp therapy or extraction were considered eligible. Only lower primary molars were analyzed because the radiographic evaluation allows a better diagnosis of the lesion in the root bifurcation region. Teeth whose initial radiographs did not allow visualization of the dental germ of the permanent successor were not included. The radiographic analysis was performed by a previously calibrated dental radiologist (kappa index = 0.89) to identify images suggestive of the rupture of the bone crypt of the permanent successor. The rupture of the follicle's bone crypt was diagnosed when the radiologist observed any discontinuity of the follicle's bone crypt in the radiographs. The lesion regression was diagnosed based on the radiographs after the endodontic treatment. When the radiolucent image located in the furca region was smaller than the initial one, it was assumed that the regression of the lesion had occurred. Patients who were under fixed orthodontic treatment or had moderate to severe fluorosis, premolars that were not fully erupted, had carious lesions or extensive restorations were not included.

Two groups were determined: Group 1 was composed of premolars whose initial radiographs showed a radiographic image suggestive of the rupture of the bone crypt, and Group 2 was composed of premolars with no rupture of the bone crypt (Figure 1).

Information on the type of intervention (extraction or pulp therapy), the patient's age at the time of intervention, and the type of material used in pulp therapy was collected from the records. A sociodemographic ~~and oral health habits~~ questionnaire was applied.

Figure 1. Flowchart of the number of participants at each stage of the study.



Dental examinations were performed by a previously calibrated examiner for the following clinical conditions: Enamel Development Defects (EDD), shape alterations, and eruption deviations (kappa index 0.904). The evaluator was unaware of which group belonged to the evaluated premolars. Examinations were conducted in a conventional dental office, under a direct light reflector and with clean and dry teeth. A flat mouth mirror (Golgran, São Paulo-SP) and a number 5 exploratory probe (Golgran, São Paulo-SP) were used. The enamel of the permanent successor premolars was assessed using the modified EDD index.[15]

In the clinical evaluation, the enamel development defects were classified as hypoplasia, when associated with reduced enamel ~~thickness~~, and hypocalcification, when associated with alterations in translucency in varying degrees. They were classified as the extent of the defect in less than 1/3 of the dental surface, greater than or equal to 1/3 and less than 2/3 of the surface or greater than 2/3. They were also classified as to the location of the defect in the gingival half, incisal half, occlusal and cuspid. Further defects and combinations are classified into additional categories of the modified DDE index. Unit defects less than 1 mm in diameter weren't recorded.[15]

In addition to the enamel development defects, shape alterations and eruption deviations were also recorded, such as dental rotation and ectopic eruption. Teeth homologous and adjacent to the permanent successors were evaluated in order to compare the results observed and to exclude any possible environmental and/or genetic influences in the occurrence of dental alterations.

Data were scanned and analyzed using the Statistical Package for Social Science software (SPSS®, version 20.0, for Windows, SPSS Inc., USA). The descriptive analysis of data was performed, and Fisher's Exact Test and Student's t-test were used for independent samples, considering significant p values < 0.05.

RESULTS

Thirty-six children, predominantly males (61.1%) with a mean age of 11.56 (± 1.95) years old, were included in the study. Forty-eight permanent teeth were evaluated, of which 20 presented with radiographic images suggestive of the rupture of the follicle bone crypt due to a radiographically visible periapical lesion, submitted to pulp therapy or an extraction. The mean follow-up time of the patients was 25.3 months, and the final periapical condition was analyzed based on the last control X-ray. The description of the radiographic analysis of the primary molar teeth can be seen in Table 1.

Table 1. The characterization of the predecessor primary molars which were radiographically evaluated.

Variables	Teeth evaluated n (%)
Tooth	
75	13 (27.1%)
74	11 (22.9%)
84	9 (18.8%)
85	15 (31.2%)
Follicle rupture	
Yes	20 (41.7%)
No	28 (58.3%)
Type of intervention	
Pulpectomy	35 (72.9%)
Extraction	13 (27.1%)
Periapical lesion involving adjacent teeth	
Yes	0 (0.0%)
No	48 (100.0%)
Age at the time of intervention	
≤ 6 years	29 (60.4%)
> 6 years	19 (39.6%)
Material used in pulp therapy	
Chloramphenicol/Tetracycline/Zinc oxide (CTZ) paste*	27 (77.1%)
Iodinated paste	2 (5.7%)
Calcium hydroxide	6 (17.2%)
Periapical condition after pulp therapy	
Lesion regression	19 (54.3%)
Lesion stabilization	13 (37.1%)
Lesion progression	3 (8.6%)

***Powder base provided by a pharmacy in proportions of 1:1:2 chloramphenicol/tetracycline/zinc oxide, respectively.**

Alterations observed in the premolars examined are described in Table 2. Alterations were observed in 10 (20.8%) teeth, 11(22.9%) adjacent to the affected primary tooth and 14 (29.1%) teeth successor to the affected primary teeth.

Table 2. Alterations observed in the premolars examined.

Variables	Premolars evaluated n (%)
Successor to treated primary	
No alterations	34 (70.8%)
Hypoplasia	2 (4.2%)
Hypocalcification	4 (8.3%)
Ectopic eruption	2 (4.2%)
Dental rotation	5 (10.4%)
Hypoplasia and hypocalcification	1 (2.1%)
Homologous to the treated primary successor	
No alterations	38 (79.2%)
Hypoplasia	1 (2.1%)
Hypocalcification	3 (6.3%)
Ectopic eruption	2 (4.2%)
Dental rotation	3 (6.3%)
Hypoplasia and hypocalcification	1 (2.1%)
Adjacent to the treated primary successor	
No alterations	37 (77.1%)
Hypoplasia	2 (4.2%)
Hypocalcification	3 (6.3%)
Ectopic eruption	1 (2.1%)
Dental rotation	4 (8.3%)
Hypoplasia and hypocalcification	1 (2.1%)

No association was found between the premolar alterations and follicle rupture due to the periapical lesion in the predecessor molar (p = 0.418). The age at the time of intervention was associated with the occurrence of alterations (p = 0.043**) (Table 3).

The higher occurrence of development defects in the first premolars (p = 0.016) and deviations in the eruption of second premolars were observed (p = 0.032) (Table 4).

DISCUSSION

Inflammatory or infectious problems resulting from pulpal necrosis in primary molars are considered risk factors for enamel development defects in premolars. [1,2,5,16,17] In this study, shape alterations, enamel development defects, and eruption deviations were present in a considerable percentage of the successor premolars of primary teeth submitted to pulp therapy or an extraction. A previous study has observed percentage of 18.1% of demarcated opacities in premolars successive to the decayed primary teeth; in contrast, the hypoplasia rate was 4.3% for the same group.[16]

In situations in which the infectious process in primary molars causes rupture of the premolar bone crypt, the most recommended treatment is the extraction of the primary tooth, whose justification is that the infectious condition is a predisposing factor for the development of enamel alterations of the successor tooth.[1,2,12,13,16] However, there is a lack of scientific evidence to support this approach. In this study, the presence of alterations in the enamel of the successor premolars was not associated with the rupture of the bone crypt. This fact may be related to the phenomena of the follicle defense, such as the formation of a fibrous and compact wall that surrounds

Table 3. Association between the occurrence of dental alterations in the permanent successor and independent variables.

Variables	Alteration	No alteration	Total	p
	n = (%)	n = (%)	n = (%)	
Bone crypt rupture				
Yes	5 (25.0)	15 (75.0)	20	0.418*
No	9 (32.1)	19 (67.9)	28	
Therapy adopted				
Pulp Therapy	11 (31.4%)	24 (68.6%)	35	0.815*
Extraction	3 (23.1%)	10 (76.9%)	13	
Age at time of intervention μ ± SD	6.71±1.43	5.91±1.11		0.043**
Treated tooth				
First primary molar	6 (30.0)	14 (70.0)	20	0.582*
Second primary molar	8 (28.6)	20 (71.4)	28	
Material used in pulp therapy				0.508*
CTZ	9 (33.3)	18 (66.7)	27	
Others	2 (25.0)	6 (75.0)	8	
Periapical condition after pulp therapy				
Lesion regression of injury	7 (36.8)	12 (63.2)	19	0.352*
Lesion persistence or progression	4 (25.0)	12 (75.0)	16	

μ = mean; SD = standard deviation; * Fisher›s exact test. ** Student›s t-test

Table 4. Association between the type of dental alteration in the permanent successor and independent variables.

Variables	Development defects		P	Eruption alterations		p
	Yes	No		Yes	No	
Bone crypt rupture						0.214*
Yes	4 (20.0)	16 (80.0)	0.429*	1 (5.0)	19 (95.0)	
No	3 (10.7)	25 (89.3)		6 (21.4)	22 (78.6)	
Treated tooth						
First primary molar	6 (30.0)	14 (70.0)	0.016*	0 (0.0)	20 (100.0)	0.032*
Second primary molar	1 (3.6)	27 (96.4)		7 (25.4)	21 (75.0)	
Material used in pulp therapy						0.299*
CTZ	3 (11.1)	24 (88.9)	0.568*	6 (22.2)	21 (77.8)	
Others	2 (25.0)	6 (75.0)		0 (0.0)	8 (100.0)	
Periapical condition after pulp therapy						
Lesion regression of injury	3 (15.8)	16 (84.2)	1.000*	4 (21.1)	15 (78.9)	0.666*
Lesion persistence or progression	2 (12.5)	14 (87.5)		2 (12.5)	14 (87.5)	
Treatment after periapical lesion permanence						
Not necessary	3 (14.3)	18 (85.7)	0.428*	4 (19.0)	17 (81.0)	0.889*
Pulp Therapy or Extraction	0 (0.0)	8 (100.0)		2 (25.0)	6 (75.0)	
Not performed	2 (33.3)	4 (66.7)		0 (0.0)	6 (100.0)	

* Fisher's exact test

it and protects it against injuries.[1,16,17] Another factor is the tolerance of the reduced enamel epithelium because when the regression of inflammation occurs, viable ameloblasts resume their activities.[16,17] However, such mechanisms may be insufficient to protect the germ from periapical inflammation chemical mediators, especially when they are at the early stages of development.[1,12,17,18]

Endodontic therapy in primary molars and the possible consequences on the enamel of developing successor teeth are a controversial subject which requires further studies. In the present study, pulp therapies performed with CTZ paste, calcium hydroxide, and iodoform paste were analyzed, and there was no association between the material used and the occurrence of enamel alterations. Studies have reported higher ectopic eruption rates of permanent teeth when predecessors were treated with zinc oxide and eugenol and/or iodoform-based pastes.[12,19,20]

Enamel development defects in the successor tooth appear to result from pre-treatment infection rather than from the procedure itself,[1,11,16,19,21] which corroborates with the results of the present study, where there was no higher prevalence of alterations in the group in which pulp therapy was performed. When well indicated and performed, pulpectomies do not seem to contribute to adverse effects on the formation of the successor tooth.[4,12,19,20] In a previous study, it was estimated that only about 10% of enamel hypoplasias were caused by necrosis of the predecessor tooth.[22] Thus, it is not surprising that the control group also demonstrated such alterations. These findings support conservative therapy in the treatment of pulpal necrosis and periapical inflammation, combined with careful clinical and radiographic monitoring.

The age group in which pulpectomies are frequently performed in primary molars is between four and six years old.[12,20] In this study, the age ranged from 3 to 10 years old. There was a correlation between an age over six years old at the time of intervention and the presence of dental alterations in the successor premolar. This association may be related to the delay of treatment, which allows greater exposure of the forming tooth to products resulting from periradicular inflammation of the primary molar. According to the literature, alteration severity depends on the stage of calcification of the developing successor germ.[12] The younger the patient, the higher the risk of permanent germ alterations.[5,22,23]

A higher occurrence of enamel development defects was observed in the first premolars. According to Kumar[8], the first primary molars present a higher number of accessory channels in the furcation region. This fact may be related to the greater permeability of the furcation area of primary molars and the consequent diffusion of bacterial toxins, inflammatory mediators and pulp degradation products, reported as a frequent cause of alterations in the development of permanent teeth.[8,16,24]

In addition, a higher occurrence of eruption deviations in second premolars was observed. The tooth location in the dental arcade and the eruption chronology may be factors associated with these deviations. The irregular rhizolysis pattern and the presence of infectious processes may also be responsible for eruption alterations.[25] Kim et al.[26] studied the effect of several local factors on the eruption of lower premolars, finding higher rotation values in second premolars when compared to first premolars. Rotations of the permanent teeth may result from pathological lesions or fluid pressure in the area of primary roots.[20] It is possible that these pressures can be directly transmitted to the permanent follicle, resulting in the rotation of the successor tooth.

The endodontic treatment of primary teeth, in addition to ceasing the infection of the periradicular tissues, favors the balance of the developing stomatognathic system. With the preservation of the primary tooth, masticatory problems, loss of space in the dental arch, eruption disorders, speech disorders, and psychological problems are avoided.[1,17] Although alterations in successor permanent teeth have been observed, they appear to be related to the pre-existing lesion and not to the treatment itself. Therefore, based on the results of the present study, there is viability for the endodontic treatment of the primary tooth, even when there is the rupture of the follicle bone crypt.

CONCLUSION

The presence of enamel alterations in premolars was not associated with the rupture of the follicle bone crypt caused by a periapical lesion in predecessor molars.

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