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ORIGINAL ARTICLE

Trauma in primary teeth and its effect on the development of permanent successors: a controlled study

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ABSTRACT

Objective: This controlled study investigated the occurrence of sequelae to permanent successors (SPS) following traumatic dental injury in primary teeth (TDIp). Additionally, this study evaluated whether TDIp is a risk factor for SPS, with a focus on an association between SPS and the child's age and type of trauma. *Materials and methods*: The trauma group (TG) consisted of permanent teeth, whose antecessors had suffered TDIp, in a population of children with complete eruption of permanent teeth. The control group (CG) consisted of permanent teeth from the same individuals, whose antecessors had not suffered TDIp. There were 214 cases of TDIp with their respective permanent teeth. In the CG 247 permanent teeth, whose antecessor had not suffered TDIp, were included. Data concerning such teeth and when TDIp occurred (in terms of the child's age) and types of SPS were collected. The chi-square test, regression logistic with generalized estimating equations (GEE) test, and risk analyses were applied to investigate the associations. *Results*: Overall, 29% of the permanent teeth in the TG presented SPS. This was compared to the development disturbances in the CG, which was 7%. The TG demonstrated the highest risk for SPS (OR, 5.388; $p = .0001$). The discolouration of enamel (37%) was the most common type of SPS found. SPS was more prone to occur in permanent teeth whose antecessors had been intruded (39%; $p < .001$). TDIp when the child was 1-year old was associated with SPS ($p < .001$). Moreover, children who had TDIp had a 4.1 times higher risk of presenting SPS. *Conclusions*: TDIp is a risk factor for the development of SPS. All types of TDIp caused SPS in this research. Additionally, the younger ages at the time of the injury and intrusions were related to SPS.

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Introduction

Traumatic dental injury in primary teeth (TDIp) may potentially interfere with odontogenesis. Depending on the site and extension of the injury, different sequelae to permanent successors (SPS) may occur, ranging from a slight disturbance in the mineralization of enamel to a sequestration of the entire tooth germ [1]. The close anatomical relationship between the apices of the primary teeth and the germ of the permanent successors explains why injuries to the primary teeth may involve the permanent dentition [2]. In the literature, the reported prevalence of SPS ranges from 20.2% [3] to 41.3% [2].

The most recent dental trauma guidelines focused on the acute management of primary dentition traumatic injury [4] and recommended a continuation of follow-up to diagnose possible SPS, which can occur as a late consequence and may negatively impact the patient's physical and psychosocial conditions, affecting his or her quality of life [5,6].

A recent systematic review [7] demonstrated that children with trauma in the primary incisors may have an increased risk of SPS as compared with children without previous trauma. Furthermore, the child's age (i.e. younger age) and type of trauma (i.e. intrusion and avulsion) are associated with more severe SPS. However, the authors of the survey in question emphasized that very few of the studies evaluated compared their data those of a control group (CG) [7]. The inclusion of a CG is crucial to verify whether other additional etiologic factors are associated with tooth disturbance in addition to TDIp. This would avoid any bias in the prevalence of dental sequelae statistics. Therefore, the present controlled study investigated whether trauma in the primary teeth is a risk factor for SPS. The association between the presence and type of SPS, along with the child's age at the time of trauma and the type of trauma, was analysed.

Methods

Ethics statement

This controlled study was conducted at the Dental Traumatology Surveillance Centre (DTSC) and was approved by the local Ethics Committee (Protocol n°. 136-12).

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Study population and data collection

The two groups, the trauma group (TG) and CG, were selected by assessing the dental records of 1500 children, with dental trauma to the primary teeth, admitted to the DTSC from March 2005 to March 2015. The CG consisted of permanent teeth from the same individuals as the TG, whose antecessors had not suffered TDIp. The examination of all children at the first dental visit after the trauma was carried out at the paediatric dentistry post-graduation clinic. As part of the clinical procedures, all post-graduation students are trained at the beginning of each year to diagnose dental trauma injuries in primary and permanent teeth and they are also trained to carry out the follow-up procedures. The training was given by the same professor in the period of this study and it was based on radiographic images and photographs. All the diagnoses are checked and the dental records were signed by the professor who was in charge of the clinical supervision on the day of the exam. It is important to point out that a child could present more than one tooth with traumatic injury. From the dental records of 1500 patients, 124 children presented a total of 214 injured primary teeth that satisfied the inclusion criteria for the TG. The CG consisted of 247 anterior teeth from the same children. Patients were included as study subjects based on the following: (i) history of trauma to primary incisors/canine and (ii) the full eruption of their permanent successors. Incomplete dental records, radiographic images and photographs of poor diagnostic quality, anterior permanent teeth with large caries lesions, restorations or traumatic injuries were excluded from this study.

Additional information and data collected from the dental records included; patient's age at the time of trauma (in years), gender (male/female), traumatized teeth (upper and lower incisors/canine of primary teeth), type of injury (luxation – concussion/subluxation/lateral luxation/extrusion/intrusion/avulsion and fractures – enamel fracture/enamel–dentin fracture without pulp exposure/enamel–dentin fracture with pulp exposure/crown-root fracture/root fracture) and SPS (discolouration of enamel – white opacity, yellow and brown, enamel hypoplasia, crown dilacerations, odontoma-like malformation, root dilacerations, partial or complete arrest of root formation and sequestration of permanent tooth germ and eruption disturbances such as late eruption). The same primary tooth could be affected by one type of injury in the periodontal tissue and up to two injuries in the hard dental tissue. Such cases were classified in the combined injuries category. At the follow-up examinations the position, colour and morphology of the permanent successors were evaluated and radiographic and photographic measures were performed on all permanent incisors. In cases of children with more than one episode of trauma in the same tooth, the data would be linked. The types of injury in primary teeth and SPS were recorded according to the criteria described by Andreasen and Andreasen [8].

Trauma group and control group

The TG consisted of erupted permanent teeth that had suffered trauma in their primary antecessor. To evaluate SPS or disturbances of non-traumatic origin, the CG consisted of permanent successors of non-traumatized primary teeth of the same child according to dental records. Therefore, anterior teeth were included in the CG, following a similar CG criteria as previously described by Andreasen and Ravn [2]. Moreover, more than one tooth could be included from the same child for each group.

To ensure that both the TG and CG were properly classified in relation to the absence, presence and type of SPS, two examiners were previously calibrated to guarantee reliability of the classification. The intra-examiner and inter-examiner agreement (intra-examiner kappa = 0.936; inter-examiner kappa = 0.945) was evaluated using photographs and radiographs. The Kappa value analysis included all different enamel defects such as discolouration enamel, hypoplasia, crown dilacerations, odontoma-like malformation, dental fluorosis, molar-incisor hypomineralization, among others. The analyses were independently performed by two expert examiners, not involved in the clinical evaluations. The radiographs were digitalized and evaluated under natural light. Photographs were taken with the patient in the dental chair declined at 90°, using a lip retractor, a digital professional camera with a 100 mm macro lens and a ring flash.

In circumstances where the same tooth presented more than one sequel, only the most severe case was use in this study. To evaluate the sequelae in permanent teeth, the criteria of Jacomo and Campos [9] were used with modifications by the current authors. These modifications included considering hypoplasia and discolouration separately, and the following decreasing order of severity: sequestration of permanent tooth germ, odontoma-like malformation, partial or complete arrest of root formation, crown dilacerations, root dilacerations, enamel hypoplasia, discolouration of enamel and eruption disturbances.

Statistical analysis

Statistical analyses were performed using SPSS software (version 17.0; SPSS, Chicago, IL). The chi-square test was used to compare categorical variables. In order to include more than one tooth per patient in the analysis, a multilevel analysis was applied, as each patient represented an individual cluster. Furthermore, the regression logistic with the generalized estimating equations (GEE) test was applied in the statistical analyses. The Odds ratio was used to assess the risk of SPS following primary dental trauma. The statistical analyses were performed by a statistical expert. The confidence interval was set at 95%.

Results

The permanent teeth, whose antecessors had experienced dental trauma, had a significantly higher risk for SPS, when compared to the permanent teeth in the CG (OR: 5.388; CI = 3.024–9.317; $p = .0001$).

The prevalence of luxations by age groups was described as follows: concussion mostly for the age groups of two ($n = 9$; 26.4%) and six ($n = 10$; 29.4%), subluxation mainly for

the age groups of two ($n = 8$; 18.1%), three ($n = 12$; 27.2%) and five ($n = 8$; 18.1%), lateral luxation mostly for the age groups of two ($n = 4$; 17.3%) and five ($n = 8$; 34.7%), extrusion mostly for the age groups of five ($n = 3$; 30%) and seven ($n = 3$; 30%), intrusion mainly for the age groups of two ($n = 14$; 31.1%) and three ($n = 10$; 22.2%), and avulsion mainly for the age groups of five ($n = 11$, 36.6%), six ($n = 6$, 20%) and seven ($n = 6$, 20%).

Table 1 shows the distribution of types (central/lateral/upper/lower incisors and canine) of injured and non-injured teeth in TG and CG. In the TG, the highest prevalence was the upper central incisors, while in the CG the upper lateral incisors were the majority.

In relation to fractures and the age of the children at the moment of the TDIp, the prevalences found were: enamel fracture mainly for the age groups of two ($n = 5$; 33.3%) and three ($n = 3$; 20%), enamel dentin fracture without pulp exposure mostly for the age groups of one ($n = 1$; 16.6%) and two ($n = 4$; 66.6%), enamel dentin fracture with pulp exposure mainly for the age groups of three ($n = 3$; 33.3%) and five ($n = 5$; 55.5%) and root fracture mostly for the age groups of four ($n = 2$; 50%) and five ($n = 2$; 50%).

Overall, 28.9% ($n = 62$) of the permanent teeth in the TG presented SPS. The prevalence of defects in the permanent teeth due to other causes and not trauma to the primary teeth in the CG was 7% ($n = 18$). Table 2 illustrates the distribution of the types of SPS evaluated by age in the TG and CG. Discolouration and enamel hypoplasia were the most common disturbances in TG (Table 2). It was found that discolouration of enamel and enamel hypoplasia accounted for 73% of the sequelae. More severe alterations were reported in the TG compared to the CG. In the CG, 5.7% ($n = 14$) of the disturbances were discolouration of enamel, so almost all of the disturbances (14 out of 17 teeth).

Table 3 shows when the SPS was related to the type of injury of its predecessor. The most serious damage was to the successor germ following intrusion ($n = 24$; 38.7%) of the primary tooth. This was followed by concussion ($n = 9$; 14%), lateral luxation ($n = 7$; 11%), avulsion ($n = 7$; 11%), subluxation ($n = 5$; 7.8%), extrusion ($n = 3$; 4.6%), enamel fracture ($n = 2$; 3%), root fracture ($n = 2$; 3%) and enamel dentin fracture with pulp exposure ($n = 1$; 1.5%). In the category of combined injuries, of the two cases that presented sequelae, one was related to intrusion and enamel dentin fracture without pulp exposure, which generated a discolouration of enamel and the other was after lateral luxation and enamel

Table 1. Distribution of teeth in trauma group (TG) and control group (CG).

Types of teeth ($n = 214$)	TG, *n* (%)	CG, *n* (%)
Central upper incisors	172 (80.0)	43 (20.0)
Lateral upper incisors	26 (19.9)	105 (80.1)
Canine upper	1 (10.0)	9 (90.0)
Central lower incisors	13 (17.6)	61 (82.4)
Lateral lower incisors	0 (0.0)	29 (100)
Canine lower	2 (100)	0 (0.0)
Total	214 (46.4)	247 (53.6)

Table 2. Prevalence of type of sequelae in permanent successors according to different age groups.

	Groups		Age (years) at time of trauma in trauma group								
Types of sequelae in permanent successors	Control group, *n* (%)	Trauma group, *n* (%)	<1 *n* (%)	1 *n* (%)	2 *n* (%)	3 *n* (%)	4 *n* (%)	5 *n* (%)	6 *n* (%)	7 *n* (%)	8 *n* (%)
No disturbance	229 (92.7)	152 (71.0)	0	2 (25)	37 (75.5)	21 (60)	13 (59.1)	43 (79.6)	22 (78.6)	11 (78.6)	3 (100)
Discolouration of enamel	14 (5.7)	24 (11.2)	0	0	5 (10.2)	3 (8.6)	2 (9.1)	5 (9.3)	6 (21.4)	3 (21.4)	0
Enamel hypoplasia	3 (1.2)	21 (9.8)	1 (100)	5 (62.5)	4 (8.1)	3 (8.6)	4 (18.2)	4 (7.4)	0	0	0
Crown dilaceration	0	2 (0.9)	0	0	0	1 (2.9)	1 (4.5)	0	0	0	0
Odontoma-like malformation	0	1 (0.5)	0	0	0	1 (2.9)	0	0	0	0	0
Root dilacerations	1 (0.4)	2 (0.9)	0	0	0	2 (5.7)	0	0	0	0	0
Partial arrest of root formations	0	1 (0.5)	0	0	0	0	0	1 (1.8)	0	0	0
Sequestration of tooth germ	0	1 (0.5)	0	1 (12.5)	0	0	0	0	0	0	0
Eruption disturbance	0	10 (4.7)	0	0	3 (6.1)	4 (11.4)	2 (9.1)	1 (1.8)	0	0	0
Total ($n = 675$)	247 (100)	214 (100)	1 (100)	8 (100)	49 (100)	35 (100)	22 (100)	54 (100)	28 (100)	14 (100)	3 (100)

Table 3. Prevalence type of sequelae in permanent successors according to different traumatic dental injuries in primary teeth (TDIp).

Types of TDIp ($n = 214$) Types of sequelae in permanent successors	Enamel fracture *n* (%)	EDF w/o pulp exposure *n* (%)	EDF with pulp exposure *n* (%)	Root fracture *n* (%)	Concussion *n* (%)	Subluxation *n* (%)	Lateral luxation *n* (%)	Extrusion *n* (%)	Intrusion *n* (%)	Avulsion *n* (%)	Combined injuries *n* (%)
Discolouration of enamel	1 (7.1)	0	0	0	5 (14.7)	0	1 (4.5)	2 (22.2)	9 (20.9)	5 (16.7)	1
Enamel hypoplasia	1 (7.1)	0	1 (14.2)	1 (25)	4 (11.8)	2 (4.7)	4 (18.1)	0	8 (18.6)	0	0
Crown dilaceration	0	0	0	0	0	0	0	0	2 (4.6)	0	0
Odontoma-like malformation	0	0	0	0	0	0	0	0	1 (2.3)	0	0
Root dilaceration	0	0	0	0	0	0	0	0	1 (2.3)	0	1
Partial arrest of root formations	0	0	0	0	0	0	1 (4.5)	0	0	0	0
Sequestration of tooth germ	0	0	0	0	0	0	0	0	1 (2.3)	0	0
Eruption disturbance	0	0	0	1 (25)	0	3 (7.1)	1 (4.5)	1 (11.1)	2 (4.6)	2 (6.7)	0
No sequelae	12(85.7)	3 (100)	6 (85.7)	2 (50)	25 (73.5)	37 (88)	15 (68.1)	6 (66.6)	19 (44.1)	23 (76.7)	4
Total, $n = 214$	14 (100)	3(100)	7 (100)	4 (100)	34 (100)	42 (100)	22 (100)	9 (100)	43 (100)	30 (100)	6

EDF: enamel–dentin fracture; w/o: without. There was no case of recurrence of dental trauma in the same tooth.

Table 4. Association of sequelae in permanent successors with types of injuries in primary teeth.

	Sequelae in permanent successors			
Types of injuries	Absent *n* (%)	Present *n* (%)	Total *n* (%)	*p* Value[a]
Enamel fracture	12 (85.8)	2 (14.2)	14 (100)	.732
EDF without pulp exposure	3 (100)	0	3 (100)	.558
EDF with pulp exposure	6 (85.8)	1 (14.2)	7 (100)	.432
Root fracture	2 (50.0)	2 (50.0)	4 (100)	.387
Concussion	25 (73.5)	9 (26.4)	34 (100)	.491
Subluxation	37 (88.1)	5 (11.9)	42 (100)	.289
Lateral luxation	15 (68.2)	7 (31.8)	22 (100)	.374
Extrusion	6 (66.7)	3 (33.3)	9 (100)	.435
Intrusion	19 (44.2)	24 (55.8)	43 (100)	.001
Avulsion	23 (76.6)	7 (23.3)	30 (100)	.180
Combined injuries	4 (66.7)	2 (33.3)	6 (100)	.554
Total	152 (70.9)	62 (29.0)	214 (100)	

EDF: enamel dentin fracture.
[a]Generalized estimating equations (GEE) test.

Table 5. Association of sequelae in permanent successors with age for the traumatic dental injuries in primary teeth (TDIp).

	Sequelae in permanent successors			
Age (years)	Absent *n* (%)	Present *n* (%)	Total *n* (%)	*p* Value[a]
<1	0	1 (100)	1 (100)	–[b]
1	2 (25.0)	6 (75.0)	8 (100)	.001
2	37 (75.5)	12 (24.5)	49 (100)	.848
3	21 (60.0)	14 (40.0)	35 (100)	.104
4	13 (59.0)	9 (40.9)	22 (100)	.336
5	43 (79.6)	11 (20.3)	54 (100)	.135
6	22 (78.5)	6 (21.4)	28 (100)	.444
7	11 (78.5)	3 (21.4)	14 (100)	.368
8	3 (100)	0 (0.0)	3 (100)	.460
Total	152 (71.0)	62 (28.9)	214 (100)	

[a]Generalized estimating equations (GEE) test.
[b]The age of up to 1-year old was excluded from the statistical tests, as the number of cases in this group ($n = 1$) was too small.

dentin fracture with pulp exposure, which led to a root dilaceration.

Table 4 shows the prevalence of SPS in comparison to the types of TDIp. The largest number of occurrences observed in this study were following intrusion. Of all the primary teeth intruded ($n = 43$), 55.8% of their permanent successors had sequelae. Intrusion was also the only injury significantly associated to the presence of SPS ($p < .001$). The other relevant prevalences of SPS after intrusion were: lateral luxation (31.8%); concussion (26%); avulsion (23%); and subluxation (11.9%).

The frequency of SPS, compared to the age at the time of TDIp, is shown in Table 5. In the only existing case of trauma prior to the child reaching 1-year old, there was a sequel. The results demonstrated that among the children who suffered trauma when they were 1-year old, 75% ($n = 6$) had some type of damage to their permanent successors. These children had the following types of trauma: intrusion ($n = 3$), lateral luxation ($n = 2$) and enamel dentin fracture ($n = 1$). The statistical analyses indicated that TDIp at the age of 1 year was significantly associated with SPS ($p < .001$). The results also indicated that the lowest prevalence of sequelae was displayed in children who were 5, 6 and 7 years old (20%, 21% and 21%, respectively). None of the children who had experienced trauma when they were 8 years of age presented SPS.

Discussion

Traumatic dental injury has been experienced by more than one billion people, while 180 million children have experienced at least one TDIp, as described in a recent study [10]. The authors evaluated 102 papers related to traumatic injuries in the permanent teeth and only 46 related to such in primary dentition [10], which amounted to less than 50% of the total studies on this subject available. TDIp is a relevant condition for the world population and necessitates that researchers continue to investigate its outcomes. In addition, primary teeth sequelae treatment could require more urgency, but SPS can affect patient quality of life with respect to posttraumatic injury [5,6]. Therefore, it is important to understand the influence of TDIp and SPS.

In this context, the correlation between TDIp and SPS has been largely investigated by several authors through observational studies [2,3,9,11,12], but most of these papers did not include a CG. In a systematic review, Lenzi et al. [7] demonstrated that TDIp is associated with SPS; however, there were confounding factors present, such as the absence of a CG that could have influenced most studies' results. At this stage of understanding, we believe that state-of-the-art dental trauma studies must include a CG in their designs.

There are other confounding etiological factors such as molar-incisor hypomineralization, amelogenesis imperfecta and fluorosis that could cause disturbances in the permanent teeth unrelated with the trauma. A controlled traumatized study design would be more suitable for use and more likely to address these confounding factors, consequently presenting more unbiased results. Due to the lack of the controlled studies on TDIp related with SPS in the literature [7], the present investigation included a CG with nontraumatized teeth.

The prevalence of SPS following TDIp has been shown to range between 20.2% [3] and 41.3% [2]. In the present study, the prevalence in the TG was 28.9%. Notably, we demonstrated that the chance of SPS in traumatized primary teeth is four times higher than that in nontraumatized primary teeth in the same child. de Amorim et al. [12] found a prevalence of 22.4% of sequelae in the permanent teeth after TDIp in a population aged up to seven years old. Despite the fact that a CG was not included by the authors; the results were similar to the present paper. Additionally, the authors found discolouration and hypoplasia as the most frequent TDIp sequelae (74.1%), corroborating with our findings.

The TG prevalence of SPS in our study and the percentage of disturbances (7%) found in the CG were in agreement with other findings in the literature. A similar result in a case-control study of a Danish population was reported by Andreasen and Ravn [2], where the CG was composed by contralateral successors of nontraumatized primary teeth. The authors concluded that nontraumatic etiology could explain less than 3% of disturbances in the permanent dentition, while TDIp explains 41%.

A significant difference between TG (52%) and CG (10%) in terms of SPS after TDIp was observed by Scerri et al. [11] in a population of Maltese children. The authors found the highest prevalence of sequelae after subluxation injuries and that the most severe sequelae happened after intrusions. In

our study, a high prevalence of enamel hypoplasia after intrusion was observed. This finding can be explained by the aspects of the intrusion, which displaces the primary teeth toward the permanent teeth germ and can lead to severe sequelae, especially in earlier permanent teeth developmental stages [13].

Andreasen and Ravn [14] reported a difference of only 13% more sequelae in the TG in a study involving children from Denmark. The authors suggested that this finding could be related to the difficulty of relying on information provided by parents or guardians, especially for CG teeth. In their study [14], the CG was composed of teeth from different groups of children. Scerri et al. [11] had a similar CG design. Both studies had a different approach from that of the present paper that included nontraumatized teeth from the same children in order to reduce the impact of the interindividual variability on the results.

It is interesting to note that the lack of caregivers' memories of minor TDIp could be also a limitation in the current study, notwithstanding the fact that the results reveal a significantly higher prevalence of SPS on the TG. The present paper was designed to consider teeth of the same child participating in the TG and the CG, which may have minimized the effects of memory bias. Notably, this design provided more reliable results with less susceptibility to bias as compared with the other CG design. This study also includes a higher number of teeth in the CG than did most other studies. A larger sample size provides more information, thus reducing the uncertainty of the results.

There was a difference between the types of SPS exhibited by the TG and the CG. Enamel defects (e.g. enamel discolouration and hypoplasia) were mostly found in the CG. Enamel discolouration could have occurred in the adjacent teeth that may be affected indirectly due to the haemorrhages of the traumatized tooth [15]. After TDIp, hypoplasia is usually seen; however, three cases of hypoplasia were detected in the CG. A possible reason for this finding is a lack of information from parents or caregivers, as the TG and CG are made up of the same children.

One unusual complication after dental trauma is root dilaceration, with one case found in our CG. Root dilaceration can occur due to idiopathic causes [15,16], and hypoplasia can be associated with genetic or environmental factors [17]; consequently, it is important that studies consider the same subject as a CG. These parallel causes were not an objective of our investigation, and can be considered a limitation of our study. More severe disturbances, such as odontoma-like malformation, sequestration of the tooth germ, and crown dilacerations, were only observed in the TG. According to Scerri et al. [11], this would indicate that these disturbances are directly correlated with TDIp.

Discolouration of the enamel and enamel hypoplasia was the most common types of SPS observed in the TG. Enamel defects are described in the literature as the most frequent SPS [3,9,11,12,18], and such predominance can also be explained by the fact that enamel discolouration or hypoplasia may be caused by minor TDIp [15]. Additionally, enamel maturation continues over an extended period of time until eruption; thus, discolouration of the enamel can occur even in older children who have not experienced any trauma [19].

Hypoplasia is observed more often following the occurrence of severe trauma to the primary tooth [15]. In this paper, hypoplasia after mild injuries such as concussion and subluxation was found with a close prevalence to discolouration of enamel. These findings may be a consequence of pulp necrosis and/or periapical inflammation after minor trauma to the primary tooth [15].

The relationship between the age of the patients at the time of injury and the occurrence of SPS indicates that the permanent tooth germ is more sensitive to injuries during its early stages of development [2]. Various authors [2,3,11,12,20] found an association between the presence of SPS and children who were very young at the time of the injury. In the present study, the same relationship was confirmed for 1-year-old children who presented increased SPS associated with trauma than as compared with children of other ages. Scerri et al. [11], Skarre et al. [21] and Ravn [22] reported that the younger the child was at the time of trauma, the more severe the SPS were.

A high prevalence of SPS following intrusion in the present study is comparable with previous findings [1,3,12,18,20,23]. In agreement with the literature [2,11,23], the following SPS are considered to be severe in nature: crown dilaceration, odontoma-like malformation, sequestration of the tooth germ, and hypoplasia; such sequelae could occur after intrusion. The high SPS after intrusion can be explained by an alveolar socket fracture or crush that increases the chance of a direct transfer of the injury to the developing permanent tooth germ [2].

The present study generally identified the existence of SPS following the occurrence of hard tissue injury in primary teeth. In some cases, there was an association of luxation and fracture in the same tooth due to the same accident, although there were few cases in which fractures without luxation occurred, which could be due to a lack of notification. In one case, the child was one year of age when the trauma occurred and so age could be a factor that influenced the development of the sequelae. This finding was also presented by Tewari et al. [24], who concluded that some fractures in the primary teeth can also cause SPS, specifically enamel discolouration and hypoplasia.

Most avulsions occurred in children older than five years of age, which may explain the presence of the low number of sequelae that followed this type of trauma. However, there was a high number of concussions that occurred in two-year-old children, in whom the permanent tooth germ is in the early stages of development, thus justifying the presence of sequelae after this type of TDIp.

The low prevalence of SPS observed after fractures, subluxation and extrusion in the present study is in agreement with the results of Andreasen and Ravn [2], who found that a minor impact only disturbs the thin soft tissue or the hard tissue barrier between the primary tooth and its permanent successor. However, our results indicate that the high prevalence of sequelae following concussion as well as the presence of SPS following all types of TDIp suggests that any

type of trauma may cause SPS, including minor injuries. Furthermore, Skaare et al. [21] also observed the existence of sequelae after concussion injury and concluded that minor luxation injuries may cause enamel defects in permanent successors.

The present study notably highlighted the importance of using radiographic exams in the follow-up of all trauma cases of primary dentition to detect any sequelae in the permanent successors early-on, especially those of a more severe nature such as changes in dental morphology and root alterations.

Conclusions

In summary, the results from this study demonstrate that TDIp is a risk factor for the development of SPS. All types of TDIp were associated with SPS in this evaluation, including minor traumas such as concussion and subluxation. The age of the child at the moment of the injury was an important factor for the development of sequelae in the permanent successors. Severe traumas, such as avulsion, in older children resulted in a lower prevalence of SPS. Additionally, the occurrence of SPS is related to younger ages at the time of the injury and intrusions. Finally, disturbances of enamel development were the most common types of SPS.

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Disclosure statement

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References

[1] Andreasen JO, Sundstrom B, Ravn JJ. The effects of traumatic injuries to primary teeth on their permanent successors. I. A clinical and histologic study of 117 injured permanent teeth. Eur J Oral Sci. 1971;79:219–283.
[2] Andreasen JO, Ravn JJ. The effect of traumatic injuries to primary teeth on their permanent successors. II. A clinical and radiographic follow-up study of 213 teeth. Eur J Oral Sci. 1971;79:284–294.
[3] Da Silva Assuncao LR, Ferelle A, Iwakura ML, et al. Effects on permanent teeth after luxation injuries to the primary predecessors: a study in children assisted at an emergency service. Dent Traumatol. 2009;25:165–170.
[4] Malmgren B, Andreasen JO, Flores MT, et al. Guidelines for the management of traumatic dental injuries: 3. Injuries in the primary dentition. Dent Traumatol. 2012;28:174–428.
[5] Arrow P. Child oral health-related quality of life (COHQoL), enamel defects of the first permanent molars and caries experience among children in Western Australia. Commun Dent Health. 2013;30:183–188.
[6] Souza JG, Martins AM, Silveira MF, et al. Impact of oral clinical problems on oral health-related quality of life in Brazilian children: a hierarchical approach. Int J Paediatr Dent. 2017;27:66–78.
[7] Lenzi MM, Alexandria AK, Ferreira DM, et al. Does trauma in the primary dentition cause sequelae in permanent successors? A systematic review. Dent Traumatol. 2015;31:79–88.
[8] Andreasen JO, Andreasen FM. Textbook and color atlas of traumatic injuries to the teeth, vol. 20. 4a ed. Copenhagen: Mosby; 2010. p. 20.
[9] Jacomo DRES, Campos V. Prevalence of sequelae in the permanent anterior teeth after trauma in their predecessors: a longitudinal study of 8 years. Dent Traumatol. 2009;25:300–304.
[10] Petti S, Glendor U, Andersson L. World traumatic dental injury prevalence and incidence, a meta-analysis—one billion living people have had traumatic dental injuries. Dent Traumatol. 2018;34:71–86.
[11] Scerri E, Gatt G, Camilleri S, et al. Morphologic and developmental disturbances of permanent teeth following trauma to primary dentition in a selected group of Maltese children. Quintessence Int. 2010;41:717–724.
[12] de Amorim LdFG, Estrela C, da Costa LRRS. Effects of traumatic dental injuries to primary teeth on permanent teeth—a clinical follow-up study. Dent Traumatol. 2011;27:117–121.
[13] Diab M, elBadrawy HE. Intrusion injuries of primary incisors. Part II: sequelae affecting the intruded primary incisors. Quintessence Int. 2000;31:335–341.
[14] Andreasen JO, Ravn JJ. Enamel changes in permanent teeth after trauma to their primary predecessors. Eur J Oral Sci. 1973;81:203–209.
[15] Arx T. Developmental disturbances in permanent teeth after following trauma to the primary dentition. Aust Dent J. 1993;38:1–10.
[16] Walia PS, Rohilla AK, Choudhary S, et al. Review of dilaceration of maxillary central incisor: a multidisciplinary challenge. Int J Clin Pediatr Dent. 2016;9:90–98.
[17] Seow WK. Clinical diagnosis of enamel defects: pitfalls and practical guidelines. Int Dent J. 1997;47:173–182.
[18] Mendoza-Mendoza A, Iglesias-Linares A, Yañez-Vico RM, et al. Prevalence and complications of trauma to the primary dentition in a subpopulation of Spanish children in southern Europe. Dent Traumatol. 2015;31:144–149.
[19] Christophersen P, Freund M, Harild L. Avulsion of primary teeth and sequelae on the permanent successors. Dent Traumatol. 2005;21:320–323.
[20] Bardellini E, Amadori F, Pasini S, et al. Dental anomalies in permanent teeth after trauma in primary dentition. J Clin Pediatr Dent. 2017;41:5–9.
[21] Skaare AB, Aas AL, Wang NJ. Enamel defects on permanent successors following luxation injuries to primary teeth and carers' experiences. Int J Paediatr Dent. 2015;25:221–228.
[22] Ravn JJ. Developmental disturbances in permanent teeth after exarticulation of their primary predecessors. Eur J Oral Sci. 1975;83:131–134.
[23] Sennhenn-Kirchner S, Jacobs HG. Traumatic injuries to the primary dentition and effects on the permanent successors a clinical follow-up study. Dent Traumatol. 2006;22:237–241.
[24] Tewari N, Mathur VP, Singh N, et al. Long-term effects of traumatic dental injuries of primary dentition on permanent successors: a retrospective study of 596 teeth. Dent Traumatol. 2018;34:129–134.