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Combination injuries 2. The risk of pulp necrosis in permanent teeth with subluxation injuries and concomitant crown fractures

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Abstract – *Background*: The reported risk of pulp necrosis (PN) is generally low in teeth with subluxation injuries. A concomitant crown fracture may increase the risk of PN in such teeth. *Aim*: To analyse the influence of a concomitant trauma-related infraction, enamel-, enamel–dentin- or enamel–dentin–pulp fracture on the risk of PN in permanent teeth with subluxation injury. *Material and Methods*: The study included 404 permanent incisors with subluxation injury from 289 patients (188 male, 101 female). Of these teeth, 137 had also suffered a concomitant crown fracture. All the teeth were examined and treated according to a standardized protocol. *Statistical Analysis*: The risk of PN was analysed separately for teeth with immature and mature root development by the Kaplan–Meier method, the log-rank test and Cox regression analysis. The level of significance was set at 5%. Risk factors included in the analysis were gender, patient age, crown fracture type, mobility and response to an electric pulp test (EPT) at the initial examination. *Results*: Teeth with immature root development: The risk of PN was increased in teeth with a concomitant enamel fracture (log-rank test: $P = 0.002$), enamel–dentin fracture (log-rank test: $P < 0.0001$), enamel–dentin–pulp fracture (log-rank test: $P < 0.0001$) and in teeth with no response to EPT at the initial examination [hazard ratio: 21 (95% confidence interval, CI: 2.5–172.5), $P = 0.005$]. Teeth with mature root development: the risk of PN was increased in teeth with an enamel–dentin fracture [hazard ratio: 12.2 (95% CI: 5.0–29.8), $P < 0.0001$], infraction [hazard ratio: 5.1 (95% CI: 1.2–21.4) $P = 0.04$] and in teeth with no response to EPT at the initial examination [hazard ratio: 8 (95% CI: 3.3–19.5), $P < 0.0001$]. *Conclusion*: A concomitant crown fracture and no response to EPT at the initial examination may be used to identify teeth at increased risk of PN following subluxation injury.

A subluxation injury is characterized by damage to the periodontal ligament with bleeding and abnormal mobility of the tooth but no displacement (1, 2). The injury may cause damage to the blood and nerve supply entering the apical foramen. A previous study has shown that the risk of pulp necrosis (PN) is rather low, especially if the root development of the tooth is immature (2). An isolated crown fracture is another type of injury that carries a low risk of PN, provided sufficient treatment is offered (3–10). However, if these two mild types of injury occur simultaneously, a new healing situation may arise. If the blood and nerve supply to the pulp is compromised because of the subluxation injury, the defence mechanisms of the pulp will become less efficient (11–14). Under these circumstances, a crown fracture may act as a pathway for bacteria that enter the pulp and cause infection (13–16). Different types of crown fracture involve different levels of communication between the oral cavity and the dentin and thereby the pulp. The access of bacteria to the dentin–pulp complex will probably not be the same in teeth with trauma-related infraction, enamel fracture, enamel–dentin fracture and enamel–dentin–pulp fracture (17, 18). It was therefore hypothesized that teeth with a subluxation injury and a concomitant crown fracture would be at higher risk of PN than teeth with a subluxation injury only because of the increased likelihood of bacterial invasion, and it was hypothesized that the risk of PN would not be the same for each of these four crown fracture types.

A recent study reported an increased frequency of PN in teeth with subluxation and crown fracture compared with teeth with a crown fracture injury only (19). However, no previous studies have analysed how these four fracture types separately affect the risk of PN in teeth having suffered subluxation injury.

The stage of root development (mature or immature) and no response to an electric pulp test (EPT) at the initial examination have previously been found to influence the frequency of PN in teeth with an isolated subluxation injury (2). These factors should also be taken into consideration when analysing possible risk factors for PN.

The aim of the present study was to analyse the influence of a concomitant trauma-related infraction, enamel-, enamel–dentin- or enamel–dentin–pulp fracture on the risk of PN in permanent teeth with subluxation injury.

Material and methods

The material includes patients treated at the Department of Oral and Maxillo-Facial Surgery, Copenhagen University Hospital, Rigshospitalet, Denmark, in the period 1971–1989.

Patients were included into the study if they fulfilled the following criteria:

1. The permanent tooth had suffered a subluxation injury defined as an injury to the tooth-supporting structures with abnormal loosening, but without displacement.
2. Tooth-specific clinical information and radiographs from the time of injury and the subsequent controls according to a standardized protocol were present.
3. Clinical photographs from the time of injury were present.
4. A follow-up period of minimum 300 days.
5. The tooth had no previous trauma.
6. No severe destruction of the crown caused by dental caries or restorations.

The standard follow-up programme included controls at 3, 6 weeks, 6 months, 1 and 5 years. The follow-up period ranged from 324 days (10.8 months) to 7750 (21.2 years) days with a median of 463 days (1.3 years).

Clinical and radiological registrations

At the time of the injury, the following parameters were registered on a special trauma chart: gender, age, cause of injury, date and time of injury, number of injured teeth, the condition of the supporting tissue and fractures of the teeth. Crown fractures were defined as infraction, enamel fracture, enamel–dentin fracture and enamel–dentin–pulp fracture, according to a modification of the WHO definition (20). For each tooth, objective clinical information from the time of injury and from follow-up examinations was recorded using a standardized form including: tooth colour, tenderness to percussion and mobility of the tooth (21).

Abnormal mobility was registered on a 0–3-point scale, where 0: no abnormal mobility; 1: facio-lingual and/or mesio-distal movement of 1 mm or less; 2: facio-lingual and/or mesio-distal movement of more than 1 mm; and 3: more than 1 mm facio-lingual and/or mesio-distal and axial mobility.

An EPT was performed using a Sirotest II® pulp tester (Siemens, Munich, Germany) (scale 0–4) placed on the incisal edge of the tooth. The test was performed at the initial examination and at follow-up examinations (21).

Horizontal and axial intraoral photographs were taken at the time of injury. Three periapical radiographs (orto-, mesio- and disto-radial/angulation) and an occlusal exposure were taken at the initial examination. At the follow-up controls, a periapical exposure was taken (21).

The stage of root development was determined by evaluation of radiographs from the initial examination and classified into one of the following six stages described by Moorrees et al. (22): 1: ¼ root formation, 2: ½ root formation; 3: ¾ root formation; 4: full root formation with wide-open apex; 5: full root formation with ½ closed apex; and 6: full root formation with closed apex. The material in the present study was divided into two subgroups: immature root development stages 1–5 and mature root development stage 6.

Pulp necrosis

PN was diagnosed if two of the following clinical signs were present:

1. Grey discoloration of the crown
2. No response to EPT after 3 months of observation
3. Periapical radiolucency

Treatment

The treatment of the crown fractures was performed according to a predefined protocol previously described (16). The majority of the teeth ($n = 320$) received no treatment of the subluxation injury, but splinting was applied in some cases. Splinting was performed either by orthodontic bands and resin or by acid-etch and a flexible temporization material (Scutan® or Protemp®3M; ESPE, Glostrup, Denmark).

Statistical methods

The material in the present study was divided into two subgroups (teeth with immature and teeth with mature root development) and analysed separately.

For teeth where PN was diagnosed in the period until 800 days after the injury, the PN onset time was approximated as the mid-point between the date of the first examination where PN was diagnosed and the date of the last examination where the tooth was not diagnosed with PN. For the remaining teeth, PN was not diagnosed in the period until 800 days after the injury (16).

The overall risk of PN was analysed using the Kaplan–Meier method (23, 24). Exact binomial confidence limits were computed for subgroups with no PN events based on the number of teeth followed for at least 1 year. Multiple Cox regression and the log-rank test were used to analyse whether the risk of PN depended on the tooth-specific crown fracture type, the mobility test and EPT outcome at the initial examination. The Cox regression analyses were further adjusted for patient gender and stage of root development (1–5) in the analysis of immature teeth and for patient gender and age in the analysis of mature teeth. Robust confidence limits were obtained taking into account that data from teeth placed in the same patients are dependent (24).

All analyses were performed with the statistical software R (R development core team, Vienna, Austria, 2010) (25).

Results

A total of 404 permanent incisors from 289 patients (188 male and 101 female) fulfilled the inclusion criteria. All teeth had suffered a subluxation injury. Furthermore, some of the teeth (n = 137) had a concomitant crown fracture (trauma-related infraction, enamel fracture, enamel–dentin fracture or enamel–dentin–pulp fracture). The teeth were separated into two groups for further analysis: 230 teeth with immature root development and 174 teeth with mature root development. The distributions of gender, patient age, number of injured teeth per patient, type of crown fracture, mobility and the response to EPT at the initial examination are given in Table 1. Data are given for the group of teeth with immature and mature root development, respectively.

A total of 231 teeth were examined and left without further treatment. Treatment delay for the remaining 173 teeth ranged from 65 min to 82 h (3.5 days) with a mean of 4.6 h. The majority of the teeth (85.6%) were treated within the first 5 h (Table 1).

Immature root development

Among teeth with immature root development, a total of 14 teeth developed PN. No teeth without crown fractures were diagnosed with PN. Therefore, crown fracture could not be included as a risk factor in the multivariate analysis. According to the log-rank test, the risk of PN was significantly higher in teeth with *enamel fracture* (P = 0.002), *enamel–dentin fracture* (P < 0.0001), and *enamel–dentin–pulp fracture* (P < 0.0001) than in teeth with no crown fracture (Table 2). Multiple Cox regression analysis showed that the risk of PN was significantly higher in teeth with no response to EPT than in teeth with a response to EPT at the initial examination [hazard ratio: 21 (95% confidence interval, CI: 2.5–172.5),

Table 2. Cox regression analysis of the risk of pulp necrosis in teeth with immature root development

	Hazard ratio	95% confidence intervals	P value
Mobility – 1st and 2nd degree	Ref. Group	–	–
Mobility – 3rd degree	5.5	1.1–27.5	0.04
Response to EPT	Ref. Group	–	–
No response to EPT	21.0	2.5–172.5	0.005
Female	Ref. Group	–	–
Male	2.3	0.7–7.8	0.20
Stage of root development (1–5)	2.8	1.4–5.5	0.003
Infraction vs no fracture[1]	–	–	NA
Enamel fracture vs no fracture[1]	–	–	0.002
Enamel–dentin fracture vs no fracture[1]	–	–	<0.0001
Enamel–dentin–pulp fracture vs no fracture[1]	–	–	<0.0001

EPT, electric pulp test at the initial examination; NA, not available.
[1]Log-rank test.

Table 1. Characteristics of patients (age and gender) and teeth (number of injured teeth in each patient, type of crown fracture, response to EPT at the initial examination, mobility at the initial examination and treatment delay) in the group of teeth with immature and mature root development

		Immature root development	Mature root development	Total
		No. of patients (%)	No. of patients (%)	No. of patients (%)
Gender	Female	64 (38.3)	37 (30.3)	101 (35.0)
	Male	103 (61.7)	85 (69.7)	188 (65.0)
Age	<20 years	167 (100.0)	102 (83.6)	269 (93.1)
	≥20 years	0 (0.0)	20 (16.4)	20 (6.9)
No. of injured teeth in each patient	One	38 (22.8)	14 (11.5)	52 (18.0)
	Two	80 (47.9)	37 (30.3)	117 (40.5)
	Three or more	49 (29.3)	71 (58.2)	120 (41.5)
		No. teeth (%)	No. teeth (%)	No. teeth (%)
Crown fracture type	None	155 (67.4)	112 (64.4)	267 (66.1)
	Infraction	2 (0.9)	8 (4.6)	10 (2.5)
	Enamel	16 (7.0)	11 (6.32)	27 (6.7)
	Enamel–dentin	48 (20.9)	31 (17.8)	79 (19.6)
	Enamel–dentin–pulp	9 (3.9)	12 (6.9)	21 (5.2)
EPT at the initial examination	No response	107 (46.5)	80 (46.0)	187 (46.3)
	Response	94 (40.9)	86 (49.4)	180 (44.6)
	Unknown	29 (12.6)	8 (4.6)	37 (9.6)
Mobility at the initial examination	1st degree	152 (66.7)	138 (79.9)	290 (72.4)
	2nd degree	54 (23.7)	23 (13.2)	77 (19.1)
	3rd degree	22 (9.6)	12 (6.9)	34 (8.5)
Treatment delay[1]	<5 h	85 (86.7)	63 (84.0)	148 (85.5)
	5–24 h	11 (11.2)	11 (14.7)	22 (12.7)
	More than 24 h	2 (2.0)	1 (1.3)	1 (1.3)

EPT, electric pulp test at the initial examination.
[1]Two hundred and thirty-one teeth were examined and left without further treatment. Treatment delay is given for the remaining 173 teeth.

P = 0.005]. Third-degree mobility (P = 0.04) and increasing stages of root development (stage 1–5) (P = 0.003) were also significantly related to PN. However, the influence of these two variables was somewhat lower than the influence of EPT (Table 2).

Table 3 shows the overall risk of PN after 12 months in teeth with each of the four different crown fracture types only and in teeth with each of the four different crown fracture types stratified by the response to EPT at the initial examination. All teeth with immature root development that developed PN gave no response to EPT at the initial examination. Figure 1 shows the risk of PN over time in teeth with immature root development and enamel–dentin fracture in relation to response of EPT at the initial examination.

Mature root development

Multiple Cox regression analysis showed that the risk of PN was significantly higher for teeth with *enamel–dentin fracture* [hazard ratio: 12.2 (95% CI: 5.0–29.8), P < 0.0001] and *infraction* [hazard ratio: 5.1 (95% CI: 1.2–21.4), P = 0.04] than for teeth with no crown fracture (Table 4). Furthermore, no response to EPT at the initial examination was associated with a significantly increased risk of PN [hazard ratio: 8 (95% CI: 3.3–19.5), P < 0.0001] in this group. Gender, age, enamel fracture, enamel–dentin–pulp fracture and mobility at the initial examination showed no statistical association with PN in teeth with mature root development (Table 4). Table 3 shows the overall risk of PN after 12 months for teeth with mature root development and for each of the four different crown fracture types only and in teeth with each of the four different crown fracture types stratified by the response to EPT at the initial examination. Figure 2 shows the risk of PN over time for teeth with mature root development and enamel–dentin fracture in relation to the response to EPT at the initial examination.

Table 3. Risk of pulp necrosis after 1 year (Kaplan–Meier estimate) for teeth with immature and mature root development in relation to type of crown fracture alone and in relation to type of crown fracture stratified by response to EPT at the initial examination

	Immature root development					Mature root development				
	Teeth at injury[1]	Teeth lost to follow up[2]	Teeth with PN[3]	Risk of PN (%)[4]	95% CI	Teeth at injury[1]	Teeth lost to follow up[2]	Teeth with PN[3]	Risk of PN (%)[4]	95% CI
No crown fracture	155	24	0	0.0	0–2.8	112	20	14	12.5	6.4–18.6
No crown fracture and response to EPT	54	11	0	0.0	0–8.2	53	12	2	3.8	0–8.7
No crown fracture and no response to EPT	75	7	0	0.0	0–5.3	55	8	12	21.8	10.6–33.1
Response to EPT Unknown	26					4				
Infraction	2	0	0	0.0	0–84.2	8	3	2	25.0	0–51.0
Infraction and response to EPT	2	0	0	0.0	0–84.2	7	3	1	14.3	0–36.5
Infraction and no response to EPT	–	–	–	–	–	1	0	1	–	–
Response to EPT Unknown	–					0				
Enamel fracture	16	2	1	6.3	0–17.4	11	0	3	27.3	2.9–51.7
Enamel fracture and response to EPT	8	2	0	0.0	0–45.9	5	0	0	0.0	0–52.2
Enamel fracture and no response to EPT	8	0	1	12.5	0–32.6	5	0	3	60.0	29.3–90.7
Response to EPT Unknown	–					1				
Enamel–dentin fracture	48	8	12	25.0	13.0–37.0	31	2	17	54.8	37.8–71.9
Enamel–dentin fracture and response to EPT	25	7	0	0.0	0–18.5	14	2	4	28.6	4.5–52.6
Enamel–dentin fracture and no response to EPT	21	1	12	57.1	40.5–73.8	15	0	13	86.7	73.9–99.4
Response to EPT Unknown	2					2				
Enamel–dentin–pulp fracture	9	0	1	11.1	0–29.4	12	0	2	16.7	0–35.9
Enamel–dentin–pulp fracture and response to EPT	5	0	0	0.0	0–52.2	7	0	0	0.0	0–41.0
Enamel–dentin–pulp fracture and no response to EPT	3	0	1	33.3	0–68.9	4	0	2	50.0	15.9–84.1
Response to EPT Unknown	1					1				

CI, confidence intervals; EPT, electric pulp test.
[1]Number of teeth at the beginning of the study.
[2]Teeth lost to follow up within the first year.
[3]Number of teeth diagnosed with pulp necrosis within the first year.
[4]Risk of pulp necrosis estimated after 1 year.

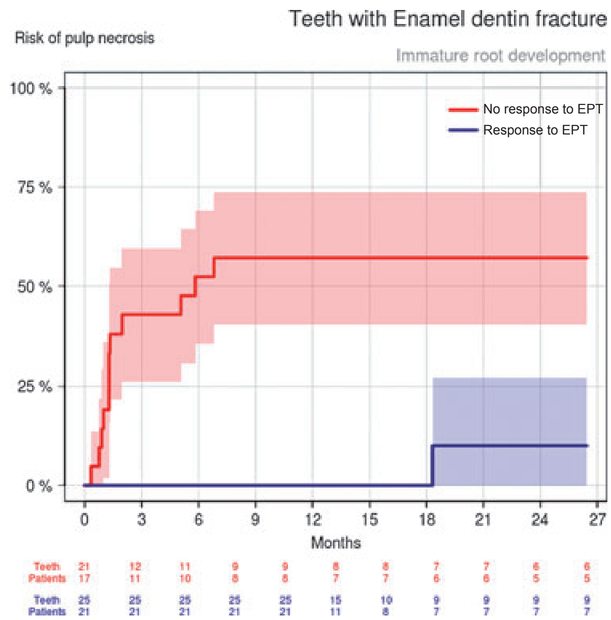

Fig. 1. The risk of pulp necrosis in teeth with subluxation, immature root development and an enamel-dentin fracture in relation to the response to the electric pulp test (EPT) at the initial examination. The shaded area represents 95% confidence interval.

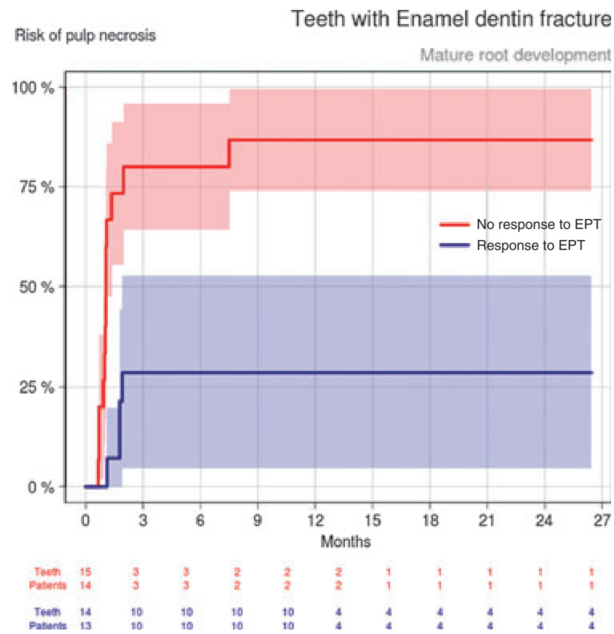

Fig. 2. The risk of pulp necrosis in teeth with subluxation, mature root development and an enamel-dentin fracture in relation to the response to the electric pulp test (EPT) at the initial examination. The shaded area represents 95% confidence interval.

Table 4. Cox regression analysis of the risk of pulp necrosis in teeth with mature root development

	Hazard ratio	95% confidence intervals	*P* value
No crown fracture	Ref. group	_	_
Infraction	5.1	1.2–21.4	0.03
Enamel fracture	2.5	0.6–10.4	0.22
Enamel–dentin fracture	12.2	5.0–29.8	<0.0001
Enamel–dentin–pulp fracture	2.8	0.7–11.9	0.16
Response to EPT	Ref. group	_	_
No response to EPT	8.0	3.3–19.5	<0.0001
Mobility – 1st and 2nd degree	Ref. group	_	_
Mobility – 3rd degree	1.8	0.5–6.5	0.36
Female	Ref. group	_	_
Male	1.6	0.7–3.8	0.25
Age < 20 years	Ref. group	_	_
Age > 20 years	2.3	0.74–7.2	0.15

EPT, electric pulp test at the initial examination.

Discussion

Trauma-related factors influencing the risk of pulp necrosis

Crown fractures

In the present study of teeth with a subluxation injury, we found a strong and highly significant association between *enamel–dentin fracture* and subsequent PN in teeth with mature and immature root development. These results support the hypothesis that healing complications are more frequently encountered in teeth with combination injuries than in teeth with subluxation only, presumably because bacteria gain easier access to the pulp when the pulp's defence mechanisms are compromised because of a subluxation injury. S*ubluxation* and *enamel–dentin fracture* are both, in principle, mild types of dental injury, each with a documented low risk of PN. However, the risk of PN is increased, and in some cases, even much increased if the two types of injury occur simultaneously.

Ravn (6) suggested that the depth and location of an enamel–dentin fracture are both factors that could influence the risk of PN. However, close inspection of his results discloses that the reported difference between small and comprehensive fractures is more likely due to differences in the treatment performed. Thus, a number of teeth with comprehensive fractures received no treatment, and the reported risk of PN was very high (54%) in these teeth. When treatment was sufficient, there was no difference in the frequency of PN between teeth with minor enamel–dentin fractures and teeth with more comprehensive enamel–dentin fractures (6). A recent study reports an increased frequency of PN in teeth with deep enamel–dentin fractures compared with a group of teeth with superficial fractures (19). However, this study did not define whether superficial fractures also included enamel fractures. We were not aware of other studies that had reported an association between fracture depth and an increased risk of PN in teeth with enamel–dentin fractures (26) and therefore decided not to incorporate the depth and location of the fracture as a variable in the present analysis.

The presence of *trauma-related infractions* significantly increased the risk of PN in teeth with mature root development. Unlike the infractions occurring spontaneously in premolars and molars (27, 28), *enamel infractions* in permanent incisors caused by trauma are usually not associated with pain. It is unknown whether fracture lines in the enamel progress into the dentin, but they are most likely arrested at the enamel–dentin junction because of the difference in the elastic modulus of enamel and dentin (29). In an *in vitro* experiment, Love (30) analysed the ability of bacteria to enter the pulp canal through infractions caused by a blow to the tooth. Intact, extracted incisors were subjected to an impact to the crown that did not cause an actual fracture. The crowns were then covered in a bacterial solution, and after 7 days, he found evidence of bacteria in the root canal of several teeth. He suggested that infractions could represent a possible way of bacterial entry. When teeth demonstrated bacterial invasion, sealing the enamel with unfilled resin prevented further infection. Despain et al. (31) performed a scanning electron microscopic investigation of replicas of the enamel of human incisors. They found infractions of varying size (between 2 and 20 μm wide) and evidence of oral debris in the largest infractions. These teeth had not been subjected to trauma. Because the size of an average oral streptococcus is proximally 0.5–0.7 μm (16), bacterial progression through an infraction line is certainly a possibility. This is further supported by the results of Ravn (32), who reported an increase in the frequency of PN in teeth with infractions from 3.5% to 34% if the teeth had a concomitant subluxation injury compared with teeth with only an infraction injury. In the present material, the infractions received no treatment, and it is possible that they allowed bacteria to gain access to the enamel–dentin junction, which would involve a risk of further progression into the pulp through dentinal tubules. In a stressful clinical treatment situation, this type of crown fracture may easily be overlooked, but the results of this study suggest that there is an additional risk to the pulp if the infraction occurs in conjunction with a subluxation injury in teeth with mature root development. Sealing of the infraction lines with unfilled resin is an easy and non-invasive treatment whose ability to prevent PN in teeth with combination injuries should be explored in future research.

Among teeth with immature root development, the univariate log-rank test showed a significantly increased risk of PN in teeth with enamel fracture and enamel–dentin–pulp fracture. However, none of the teeth that developed PN responded to EPT at the initial examination (Table 4). The effect of EPT cannot be distinguished from the effect of the crown fracture in these teeth.

When multivariate analysis was performed in teeth with mature root development, no significant association was found between *enamel fractures* and later development of PN. This observation is in accordance with the results reported by Ravn (7). Although the overall risk of PN was increased in teeth with enamel fracture, stratification for the results of the EPT made clear that all the teeth that developed PN in this group had no response to EPT at the initial examination.

Likewise, no significant association was found between fractures with pulp exposure and the subsequent development of PN. Many of these teeth responded to EPT (n = 12) at the initial examination. This indicates that the effect on the neuro-vascular supply was small and that the treatment, which consisted of pulp capping and a temporary crown, offered a sufficient seal against bacteria from the oral cavity. The majority of the patients (85.5%) were treated within the first 5 h after the trauma had occurred.

Electric pulp test at time of injury

The EPT is a measure of the sensory nerve conductivity of the pulp (33). The sensory nerves and the blood vessels enter the pulp through the apical foramen and they are closely related. It is therefore likely that when the sensory nerves are affected by the trauma, then the blood supply is affected as well. Both the function of sensory nerves and a sufficient blood supply are important for the pulp's defence mechanisms (12). No response to EPT at the initial examination may therefore be an indication of possible damage to the pulp which will weaken the pulp's defence against bacterial infection. In the present study, we found a strong association between no response to EPT at the initial examination and the development of PN in teeth with immature as well as mature root development. Performed at the time when the patient is first treated for the traumatic injury, the pulp sensibility test can therefore provide important information about the prognosis of the pulp in teeth with subluxation injury.

Abnormal mobility at time of injury

Increased tooth mobility suggests a heightened level of damage to the periodontal ligament and the blood vessels and nerves entering the apical foramen. It was therefore expected that third-degree mobility (horizontal *and* axial movement) at the time of the injury would be associated with an increased risk of PN. In this material, this was only evident for immature teeth.

Patient-related factors affecting the risk of pulp necrosis

Age

As the majority of dental traumas happen in children, the literature offers little information on the consequence of higher age on the healing of dental trauma. In the present study, we found no statistical association between increasing age and PN. This may be due to the low number of older patients in the study (Table 1).

Stage of root development

Because of the different healing potential for teeth with mature and immature root development previously reported (2), teeth with immature and mature root development were analysed separately.

Within the group of teeth with immature root development, the risk of PN increased with increasing root development (stage 1–5). As the root develops, the contact area between the pulp and the periodontium decreases, and this will affect the healing capacity of the pulp. This finding is in accordance with findings reported in previous studies (2, 19).

Treatment-related factors affecting the risk of pulp necrosis

This study was not designed to measure the effect of different treatment strategies as all patients within each crown fracture group were treated according to similar principles. The potential variance that could arise if treatment-related factors had affected the pulp healing was thereby minimized.

A total of 231 teeth did not require treatment at the initial visit. The majority of the remaining 173 teeth received treatment within the first 5 h after the trauma had occurred. Treatment delay was therefore not included as a risk factor in the analysis. However, regression analysis was performed on a subset of the population namely teeth with mature root development which received treatment (n = 75). This showed a significantly higher risk of PN if the treatment delay exceeded 5 h [hazard ratio 4.2 (95% CI: 1.6–10.7) P = 0.003]. Further analysis of the effect of treatment delay should be performed in future studies. Based on the result of this study, it is recommended that crown fractures be restored and enamel infractions sealed as soon as possible to reduce the risk of PN in teeth with subluxation.

Conclusion

The presence of a concomitant crown fracture or no response to sensibility test at the initial examination significantly increased the risk of PN in teeth with subluxation injury. These two risk factors may therefore be used to identify teeth at increased risk of subsequent PN following subluxation injury. Based on the results of the present study, it is recommended that crown fractures in subluxated teeth be restored and enamel infractions sealed as soon as possible to reduce the risk of PN.

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