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Combination injuries 1. The risk of pulp necrosis in permanent teeth with concussion injuries and concomitant crown fractures

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Abstract – *Background*: The reported risk of pulp necrosis (PN) is low in teeth with concussion injuries. A concomitant crown fracture may affect the risk of PN. *Aim*: To analyze the influence of a crown fracture (with and without pulp exposure) on the risk of PN in teeth with concussion injury. *Material*: The study included 469 permanent incisors with concussion from 358 patients (226 male, 132 female). Among these, 292 had a concomitant crown fracture (70 with and 222 without pulp exposure). All teeth were examined and treated according to standardized protocol. *Statistical analysis*: The risk of PN was analyzed by the Kaplan–Meier method and Cox regression. Risk factors included in the analysis: gender, age, stage of root development, type of crown fracture, and response to electric pulp test (EPT) at the initial examination. The level of significance was set at 5%. *Results*: The risk of PN was low in teeth with immature root development [1.1%, 95% confidence intervals (CI): 0–3.4]. The following factors significantly increased the risk of PN in teeth with mature root development: crown fracture without pulp exposure [hazard ratio 4.1 (95% CI: 1.4–11.9), $P = 0.01$] and no response to EPT at the initial examination [hazard ratio 30.7 (95% CI: 7.7–121), $P < 0.0001$]. The overall risk of PN increased from 3.5% (95% CI: 0.2–6.8) to 11.0% (95% CI: 5.2–16.7) when a concomitant crown fracture without pulp exposure was present. If the tooth had both a crown fracture and gave no response to EPT, the risk further increased to 55.0% (95% CI: 34.3–75.8). *Conclusion*: No response to EPT at the initial examination or a concomitant crown fracture significantly increased the risk of PN in teeth with concussion injury and mature root development. If both risk factors were present there was a synergetic effect.

Pulp necrosis (PN) is a frequent complication after a luxation injury. PN can occur either as ischemic sterile necrosis (infarction) caused by disruption of the blood supply at the apical foramen or as an infection-related liquefactive necrosis (1). The extent of the damage to the blood and nerve supply to the pulp depends on the severity of the luxation injury (2). It may range from minor injury such as local bleeding, stretching, or compression of the nerves and blood vessels in case of a concussion or a subluxation to complete disruption of the blood and nerve supply to the pulp which is likely to occur in a lateral luxation. If bacteria gain access to an injured or ischemic pulp, the healing will be affected. Contamination of the root canal has been reported as the principle cause of failure of a pulp to revascularize after ischemic necrosis (3).

Crown fracture with exposed dentinal tubules has been proposed as a way for bacteria to gain access to the pulp (4, 5). An experimental study in humans has shown that teeth with vital pulp were more resistant to bacterial invasion into the dentinal tubules than teeth with non-vital pulp (6). This can probably be explained by the defense mechanisms of the healthy pulp which protects the tissue against bacterial invasion (7–10). Hence, several clinical studies have reported a low frequency of pulp necrosis (0–3%) in teeth with an isolated crown fracture that does not involve the pulp (11–15). However, if the blood and nerve supply to the pulp is compromised or completely disrupted, the defense mechanisms may be less efficient (9, 15). Crown fractures may then act as a pathway for bacteria to enter the pulp and cause infection. It was therefore hypothesized that the risk of pulp necrosis was higher in teeth with combination injuries – i.e. teeth with a luxation and a concomitant crown fracture injury – than in teeth with a luxation injury only.

A combination injury caused by dental trauma represents a very complex healing scenario and many factors can influence the possibility of successful pulp healing. To analyze how a concomitant crown fracture affects the risk of PN in teeth with luxation injuries, it is

necessary to analyze each luxation type separately, and, if possible, incorporate relevant patient-, trauma-, or treatment-related factors in the analysis. In a series of three articles, the complex healing scenario of teeth with combination injuries has been analyzed.

In the present study, it was analyzed how pulp healing was affected by the occurrence of two simultaneous, minor injuries: a crown fracture and a concussion injury. In the literature, the reported frequency of PN is low in teeth that have suffered concussion injury (2). The stage of root development (mature or immature) has been found to influence the risk of PN (2). For teeth with a crown fracture, the risk of PN may be influenced by the type of crown fracture and the treatment performed (16, 17). Furthermore, the risk of PN in teeth with a crown fracture has been shown to be increased if there was no response to a sensibility test at the initial examination (18). These factors should therefore also be taken into consideration when assessing the prognosis of the pulp.

The aim of the present study was to analyze the influence of a concomitant crown fracture (with and without pulp exposure) on the risk of PN in teeth with concussion injury.

Material and methods

The material included patients treated at the Department of Oral and Maxillo-Facial Surgery, Copenhagen University Hospital, Rigshospitalet, Denmark in the period from 1972 to 1990.

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

1 The permanent tooth had suffered a *concussion* injury defined as an injury to the tooth-supporting structures without abnormal loosening or displacement, but with a marked reaction to percussion.

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 program included controls at 3, 6 weeks, 6 months, 1, and 5 years. The follow-up period ranged from 338 to 8045 days (22.0 years) with a median of 420 days (1.2 years).

Clinical and radiological registrations

At the time of the injury, the following parameters were registered on a special trauma chart: gender, patient 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 classified as trauma-related infraction, enamel fracture, enamel-dentin fracture, and enamel-dentin-pulp fracture, according to a modification of the WHO definition (19). The four types of crown fracture were grouped as fractures with and without pulp exposure. For each tooth, objective clinical information from the time of injury and from follow-up examinations was recorded using a standardized form including: tooth color, tenderness to percussion, and mobility of the tooth (20).

Electric pulp test (EPT) was performed using a Sirotest II® pulp tester (Siemens, Munich, Germany) (scale 0–4) placed on the incisal edge of the tooth (20). The test was performed at the initial examination and at follow-up examinations.

Horizontal and axial photographs were taken at the time of injury. Three periapical radiographs (ortho-, mesio-, and distoradial/angulation) and an occlusal exposure were taken at the initial examination. At the follow-up controls, a periapical exposure was taken (20).

The stage of root development was determined by evaluation of radiographs from the initial examination. The teeth were divided into two groups. Immature root development: the root development was incomplete and/or the apex was not fully formed. Mature root development: the tooth had full root formation with a closed apex. Teeth with immature and mature root development were analyzed separately.

Treatment

Specially trained oral surgeons at the Department of Oral- and Maxillo-Facial Surgery, Copenhagen University Hospital, Rigshospitalet, Denmark performed the initial treatment according to the following guidelines:

1 Infractions received no treatment.

2 Enamel fractures were left without coverage, but sharp enamel edges were ground.

3 Enamel-dentin fractures were treated with coverage of exposed dentin with hard-setting calcium hydroxide cement (Dycal®; DeTrey Dentsply, Addlestone, Surrey, UK) and a temporary steel crown (cemented with Bondalcap®; Vivadent, Schaan, Liechtenstein, Durelon®; 3M ESPE, Seefeld, Germany or Zinc oxide-eugenol-based compound, IRM®(DentsplyCaulk, Milford, DE, USA)) or a temporarization material (Scutan® or Protemp®; 3M ESPE).

4 In enamel-dentin-pulp fractures, the exposed pulp was rinsed with Tubulicid® (Global Dental Products, Baltimore, MD, USA), and pulp capping was performed using calcium hydroxide (Calasept®; Nordiska Dental, Ängelholm Sweden) and a temporary steel crown (cemented with Bondalcap®; Vivadent, Durelon®; 3M ESPE or Zinc oxide-eugenol-based compound, IRM®) or a temporarization material (Scutan® or Protemp®; 3M ESPE). Permanent treatment was performed after approximately 3 months when a hard tissue bridge was observed.

5 The patients were referred to private dentists or municipal school dentistry for definitive composite build-up of the fractured tooth.

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.

Statistical methods

For teeth where PN was diagnosed in the period until 800 days after the injury, we approximated the PN onset time as the midpoint between the date of the first examination where PN was diagnosed and the date of the last examination where the respective tooth was not diagnosed with PN.

The overall risk of PN was analyzed using the Kaplan–Meier method (21, 22). Changes of the risk of PN due to concomitant crown fracture without pulp exposure were assessed by multiple Cox regression (mature root development only) where further risk factors were gender, age, and EPT outcome at the initial examination. Robust confidence limits and *P* values were obtained to account for the dependencies of teeth placed in the same patients (22). Exact binomial confidence limits were computed for subgroups with no PN events based on the number of teeth followed for at least 1 year. All analyses were performed using the statistical software R. (R development core team. Vienna. Austria. 2010) (23).

Results

A total of 469 permanent incisors from 358 patients (226 male and 132 female) fulfilled the inclusion criteria. All teeth had suffered a concussion injury. Furthermore, some of the teeth ($n = 292$) 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: immature root development ($n = 169$) and mature root development ($n = 300$). The distributions of gender, patient age, number of injured teeth per patient, type of crown fracture, and the response to EPT at the initial examination are given in Table 1.

About half of the teeth ($n = 206$) teeth were examined, but received no further treatment at the initial visit. Treatment delay for the remaining teeth ranged from 45 min to 113 h (4.7 days) with a mean of 5.2 h. The majority of the teeth (85%) that received treatment appeared at the emergency clinic within the first 5 h after the trauma had occurred (Table 1).

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

	Immature root development	Mature root development	Total
	No. patients (%)	No. patients (%)	No. patients (%)
Gender			
Female	41 (30.6)	91 (40.6)	134 (37.4)
Male	93 (69.4)	133 (59.4)	224 (62.6)
Age (years)			
<20	134 (100.0)	183 (81.7)	317 (88.6)
≥20	0 (0.0)	41 (18.3)	41 (11.4)
No. of injured teeth in each patient			
One	42 (31.3)	34 (15.2)	76 (21.2)
Two	61 (45.5)	90 (40.2)	151 (42.2)
Three or more	31 (23.1)	100 (44.6)	131 (36.6)
	No. teeth (%)	No. teeth (%)	No. teeth (%)
Crown fracture type			
No crown fracture	62 (36.7)	115 (38.2)	177 (37.7)
Infraction	4 (2.4)	38 (12.8)	42 (9.0)
Enamel	19 (11.2)	18 (6.0)	37 (7.9)
Enamel-dentin	62 (36.7)	81 (27.0)	143 (30.5)
Enamel-dentin-pulp	22 (13.0)	48 (16.0)	70 (14.9)
EPT at the initial examination			
No response	26 (15.4)	39 (13.0)	65 (13.9)
Response	135 (79.9)	247 (82.0)	382 (81.2)
Unknown	8 (4.7)	15 (5.0)	23 (4.9)
Treatment delay[1] (h)			
<5	88 (92.6)	135 (80.4)	218 (84.8)
5–24	6 (6.3)	28 (16.6)	34 (12.9)
More than 24	1 (1.1)	5 (3.0)	3 (2.3)

EPT, electric pulp test.
[1]206 teeth were examined and left without further treatment. Treatment delay is given for the remaining 263 teeth.

Immature root development

Among teeth with immature root development, only one tooth developed PN. This tooth had an enamel-dentin fracture and gave no response to EPT at the initial examination. Hence, the overall risk of PN after 1 year for teeth with immature root development and concomitant crown fracture was very low [1.1%, 95% confidence intervals (CI): 0–3.4] (Table 2). No further analysis was performed.

Mature root development

The results of the multivariate analysis of teeth with concussion and mature root development are shown in Table 3. No teeth with enamel-dentin-pulp fracture developed PN during follow up. This group of teeth was therefore not included in the regression analysis. The presence of a crown fracture without pulp exposure significantly increased the risk of PN [hazard ratio 4.1 (95% CI: 1.4–11.9), $P = 0.01$]. No response to the EPT at the initial examination showed the strongest association with subsequent development of PN [hazard ratio $= 30.7$ (95% CI: 7.7–121.6) $P < 0.0001$]. Gender and age did not significantly affect the risk of PN in teeth with mature root development ($P > 0.05$).

Table 2 shows the overall risk of PN after 12 months in teeth with crown fracture (with and without pulp exposure) and in teeth with and without crown fracture stratified by the result of the EPT at the initial examination. The overall risk of PN increased from 3.5% (95% CI: 0.2–6.8) to 11.0% (95% CI: 5.2–16.7) when a concomitant crown fracture without pulp involvement was present. However, if both significant risk factors were present (concomitant crown fracture without pulp exposure *and* no response to EPT), then the risk increased to 55.0% (95% CI: 34.3–75.8). No teeth with crown fractures exposing the pulp developed PN. Figure 1 shows the development of the risk of PN over time in teeth with no crown fracture in relation to the response to EPT at the

Table 2. 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 for type of crown fracture stratified by response to electric pulp test (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	62	8	0	**0**	(0–6.6)	115	31	4	**3.5**	(0.2–6.8)
No crown fracture and response to EPT	36	4	0	**0**	(0–10.9)	96	28	0	**0**	(0–5.3)
No crown fracture and no response to EPT	20	4	0	**0**	(0–20.6)	17	3	4	**23.5**	(4.4–42.7)
Response to EPT unknown	6					2				
Crown fracture *without* pulp exposure	85	20	1	**1.1**	(0–3.4)	137	20	15	**11.0**	(5.2–16.7)
Crown fracture without pulp exposure and response to EPT	77	19	0	**0**	(0–6.1)	114	18	4	**3.5**	(0–7.6)
Crown fracture without pulp exposure and no response to EPT	6	1	1	**16.7**	(0–41.5)	20	2	11	**55.0**	(34.3–75.8)
Response to EPT unknown	2					3				
Crown fracture *with* pulp exposure	22	1	0	**0**	(0.2–11)	48	8	0	**0**	(0.8–8.1)

CI, confidence intervals; PN, pulp necrosis.
[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.

Table 3. Cox regression analysis of the risk of pulp necrosis in teeth with mature root development (teeth with enamel-dentin–pulp fractures were excluded)

	Hazard ratio	95% confidence intervals	*P* value
No crown fracture	Ref. group	–	–
Crown fracture without pulp exposure	4.1	(1.4–11.9)	0.01
Response to EPT	Ref. group	–	–
No response to EPT	30.7	(7.7–121.6)	<0.0001
Male	Ref. group	–	–
Female	0.7	(0.3–1.9)	0.49
Age <20 years	Ref. group	–	–
Age >20 years	1.4	(0.3–5.9)	0.63

EPT, electric pulp test at the initial examination.

initial examination. Figure 2 shows the development of the risk of PN over time in teeth with a crown fracture without pulp exposure in relation to the response to EPT at the initial examination.

Discussion

Trauma-related factors affecting the risk of pulp necrosis

Crown fractures

The aim of the present study was to analyze the effect of a concomitant crown fracture (with or without pulp exposure) on the risk of PN in teeth with a mild luxation injury such as a concussion. We found that the risk of PN was significantly increased in teeth with mature root development when a crown fracture without pulp exposure occurred in combination with a concussion injury. A concussion injury is defined as an injury to the tooth-supporting structures without abnormal loosening or displacement, but with a marked reaction to percussion. Owing to the limited damage to the periodontal tissue in teeth with concussion injury, it is likely that PN was caused not by complete ischemia, but by infection. The result therefore supports our hypothesis that there is an increased probability of bacterial penetration through exposed dentinal tubules when the defense mechanism of the pulp is affected by the concussion injury.

In the multivariate analysis, infractions, enamel fractures, and enamel-dentin fractures were grouped as teeth with crown fractures without pulp exposure. Due to the low number of teeth with PN found in this study, it was not possible to analyze the individual types of crown fracture separately.

The outcome for the 70 teeth with fractures exposing the pulp (enamel-dentin-pulp fracture) was excellent. Different authors report very different frequencies of PN in teeth with such fractures (15, 24–26), ranging from 0 (15) to 57% (24). This large variation may be explained by differences in the treatment performed and differences in the time that elapsed from the trauma occurred until treatment was performed (17). Furthermore, if a patient does not appear for treatment until days after the

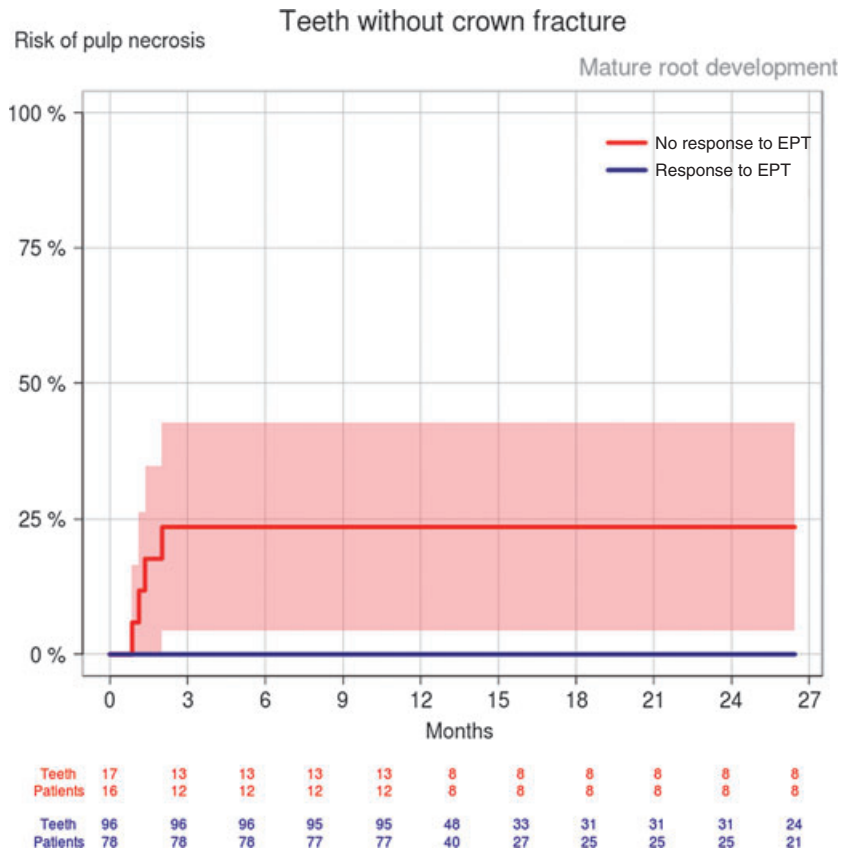

Fig. 1. The risk of pulp necrosis over time in teeth with concussion, mature root development and no crown fracture. Red: Teeth which showed no response to electric pulp test (EPT) at the initial examination. Blue: Teeth which responded to EPT at the initial examination. The shaded area represents 95% confidence intervals.

trauma, the diagnosis may be incorrect because symptoms of concussion and subluxation may have subsided. In the present study, no teeth with crown fracture with pulp exposure developed PN. Only two teeth with enamel-dentin-pulp fracture showed no response to the EPT at the initial examination. This indicates that for the majority of these teeth, the effect on the pulp was minor 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%) were treated within the first 5 h after the trauma had occurred.

Electric pulp test at the initial examination

No response to EPT at the initial examination was the risk factor that was most strongly associated with subsequent PN (Table 3). EPT measures the sensory nerve conductivity of the pulp. A response to the test indicates that the nerve fibers are functioning, but it is important to emphasize that the EPT does not measure the extent of blood flow and thereby the vitality of the pulp (27). Concussion is a mild luxation injury and it is possible that compression or stretching of the nerve fibers may temporarily affect the sensibility of the tooth (28). The majority of the nerves innervating the dental pulp are sensory nerves. When stimulated, they release vaso-regulating neuro-peptides (8). The nerves of the pulp therefore play an important role in the inflammatory reaction and, thereby, in the healing and defense of the pulp as they induce an increased blood flow to the injured area as well as an increased vascular permeability (8). Furthermore, when the nerves are affected, it is also possible that the blood supply is affected because blood vessels and sensory nerves enter the root canal in close proximity. Hence, no response to EPT at the initial examination may reflect damage to the pulp caused by the trauma.

The use of EPT at the initial examination of traumatized teeth is controversial. The test requires the patient's cooperation and it may sometimes be difficult to perform if the patient is very stressed and upset. In this study, it was possible to perform the test in 95% of the traumatized teeth and the results indicated a strong and highly significant association between no response to EPT at the initial examination and subsequent development of PN in teeth with mature root development. This result is supported by a previous study by Zadik et al. (18) who concluded that the pulp prognosis was unfavorable when there was no response to EPT at the initial examination in teeth with a crown fracture injury. If it is possible to perform the test at the initial examination we may obtain information about the condition of the pulp at the day of the trauma as well as the risk of subsequent healing complications in teeth with a concussion injury.

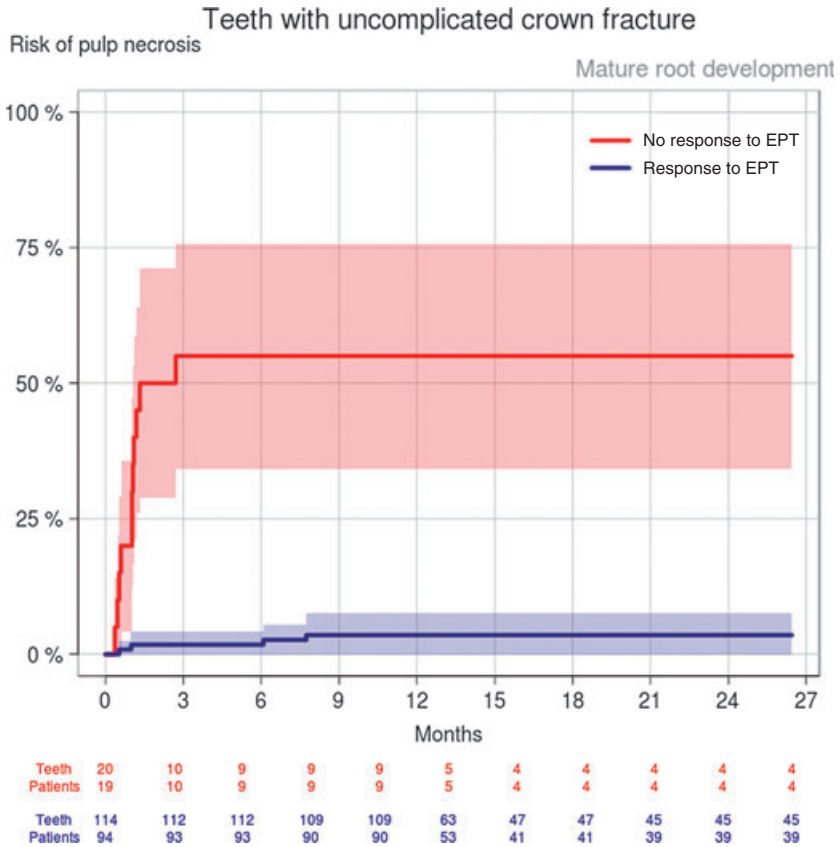

Fig. 2. The risk of pulp necrosis over time in teeth with concussion, mature root development and crown fracture (without pulp exposure). Red: Teeth which showed no response to electric pulp test (EPT) at the initial examination. Blue: Teeth which responded to EPT at the initial examination. The shaded area represents 95% confidence.

In teeth with mature root development, the severity of the trauma expressed in terms of no response to EPT was the most important factor influencing the risk of PN. In addition, the presence of a concomitant crown fracture indicated an even higher risk of PN. A tooth with both a crown fracture and no response to EPT carried the highest risk of PN among teeth in this material (Fig. 2). To identify teeth at risk of subsequent PN following concussion injury, both risk factors (crown fracture and no response to EPT) should therefore be considered.

Patient-related factors affecting the risk of pulp necrosis

Stage of root development

Andreasen et al. (2) concluded that the stage of root development was one of the most important factors affecting pulp healing. The influence of a concomitant crown fracture may not be similar in teeth with mature and immature root development. To minimize the variance of our material it was therefore decided to analyze teeth with mature and immature root development separately.

The patient's age

No association was found between increasing age and PN in the multivariate analysis. This may be due to the low number of older patients in the study (Table 1).

Treatment-related factors affecting the risk of pulp necrosis

Nearly half of the teeth included in the study (n = 206) did not require treatment at the initial visit. The majority of the remaining teeth (85%) received treatment within the first 5 h after the trauma. The effect of treatment delay on the risk of PN was therefore not included in the analysis. Furthermore, the 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.

Discussion of statistical methods

It is not possible to determine the exact onset times of pulp necrosis. It was only known that PN occurred between the dates of two consecutive examinations. Therefore, the midpoint approach was used were it was assumed that the midpoint between two examinations was the true onset time of pulp necrosis. This may have introduced a small bias in the results.

The Cox regression model assumes that the effects of the predictor variables on the risk of PN are constant within the considered time period (proportional hazards). This assumption becomes more problematic the longer the time period. It was therefore decided to limit all analyses to the first 800 days after the trauma. This

decision was supported by the fact that PN usually occurs within the first 2 years after the trauma. PN diagnosed more than 2 years after the trauma occurred is most likely not directly related to the trauma, but rather to breakdown of restorations or caries. Furthermore, by restricting the analysis to this time period, we avoided a complex discussion about follow-up compliance at times later than 2 years after the trauma. Thus, a necessary condition for all survival analyses performed is that the patients lost to follow up have the same risk of PN as those who attend the follow-up examinations.

Conclusion

In conclusion, the risk of pulp necrosis due to a concussion injury was generally low. However, no response to pulp sensibility test at the initial examination or the presence of a concomitant crown fracture without pulp exposure significantly increased risk of pulp necrosis in teeth with mature root development. If both risk factors were present there was a synergetic effect. These two risk factors may be used to identify teeth at increased risk of subsequent pulp necrosis following concussion injury.

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