The Cracked Tooth: Histopathologic and Histobacteriologic Aspects

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Abstract

Introduction: The diagnosis and treatment planning of cracked teeth depend on the understanding of how cracks affect the surrounding tissues. This study evaluated the dentin and pulp conditions in teeth affected by cracks and attrition. **Methods:** Specimens under investigation included 12 cracked posterior teeth and 8 teeth with severe attrition. These teeth were obtained consecutively in a private practice and were extracted for reasons not related to this study. Teeth were processed for histopathologic and histobacteriologic analyses. **Results:** Cracks were histologically detected in all specimens, including the teeth with severe attrition. The cracks in all teeth were colonized by bacterial biofilms. One tooth showed several craze lines in the enamel, one of which reached dentin to a shallow depth. In some teeth, the crack ended in the dentin. Dentinal tubules were invaded by bacteria, especially when the crack extended perpendicularly into the dentin. Severe accumulations of inflammatory cells were present in the pulp zone subjacent to tubules involved with the crack. In many cases, the crack extended to the pulp, leading to reactions with intensities ranging from acute inflammation to total pulpal necrosis. Symptoms occurred in most cases in which the pulp was affected. In some cases, polymorphonuclear neutrophils were seen migrating from the pulp into the crack space and facing the bacterial biofilm located therein. Severe pulp reactions were also observed when the crack extended to the pulp chamber floor. **Conclusions:** Cracks are always colonized with bacterial biofilms. The pulp tissue response varies according to the location, direction, and extent of the crack. *(J Endod 2015;41:343–352)*

Key Words

Bacterial biofilm, cracked teeth, dentin, pulp inflammation, tooth fracture

A cracked tooth can be defined as an incomplete fracture of the tooth structure that can progress to affect the pulp and periodontal ligament. Possible causes for this condition include repetitive heavy mastication, occlusal prematurities, physical trauma, resorption, caries, weakened teeth, iatrogenic dental treatment, and damaging parafunctional or factitial habits (1–6). Clinically, the presence and extent of cracks in teeth can be very difficult to objectively determine. For diagnosis and treatment planning, there are many parameters that should be taken into account including the presence of symptoms; pulp status (reversible pulpitis, irreversible pulpitis, or necrosis); periodontal status; direct visualization; and the history of restorative procedures, caries, previous dental trauma, or parafunctional habits. Magnification, transillumination, and staining with a disclosing solution are useful diagnostic tools; however, even though a crack may be detected, its extent is hard to guess.

Recommending appropriate treatment for teeth with cracks of unknown depth is a complicated task. Cuspal reinforcement with a crown (7, 8) or bonded restoration (7, 9, 10) has been suggested for cracked teeth with vital pulps, with the understanding that the prognosis may be unpredictable. If the crack line progresses deep in the tooth structure, the pulp may become affected (11, 12). In addition, the crack line can extend further and reach the periodontal tissues to cause a bony dehiscence, narrow and deep periodontal pocket, and/or extensive periradicular bone resorption (13, 14). It has been suggested that should a crack extend through the pulp chamber floor or apically to the level of the alveolar bone crest, the prognosis is considered poor, and tooth extraction should be considered (5, 12).

Although it has been historically considered that cracks occur mostly in teeth with large or inadequate restorations, a more recent study (15) revealed the presence of cracks in about 60% of the evaluated molar teeth with no restorations. In another investigation (5), 27 nonvital teeth with minimal or no restorations were evaluated in which pulp necrosis was thought to have occurred secondary to longitudinal fractures. Micro–computed tomographic scans revealed that 100% of these teeth had fractures that extended from the coronal tooth structure into the pulp and advanced toward the lateral root surface. Because the fracture was thought to have been the cause of pulp necrosis, these types of fracture were referred to as "fracture necrosis" (5).

Although there are many articles discussing the diagnosis, treatment considerations, and prognosis of teeth affected by cracks, investigations regarding the dentin and pulpal responses to cracks as well as the microbiological aspects of these conditions remain scarce in the literature. There are some studies on the morphologic and histobacteriologic aspects of vertical root fractures (13, 16), but, to the best of our knowledge, there are no consistent reports on the histopathology and histobacteriology of cracked teeth. The only histomorphologic description of dental cracks relates to anterior animal and human teeth affected by severe attrition, which dates back to the early 1970s (17–19). More recently, 1 study used scanning

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electron microscopy to examine cracks in vital teeth and revealed the presence of bacterial colonies in all of the observed fractures (20). Understanding the tissue reactions to cracks is crucial for the diagnosis, treatment planning, and development of better therapeutic strategies for managing these conditions. The present study was intended to histopathologically and histobacteriologically evaluate the effects of cracks on the pulp and surrounding dentin in posterior teeth as well as teeth affected by severe attrition.

Materials and Methods

Twenty human teeth were obtained consecutively in a private practice operated by 1 of the authors (DR) over a period of 15 years (Table 1). Twelve of these were posterior teeth (10 molars and 2 premolars) presenting with a clinical diagnosis of "reversible pulpitis," "irreversible pulpitis," or "pulp necrosis." Enamel and dentin cracks of varying severities were diagnosed on the basis of clinical examination using a magnifying device (EyeMag Pro 4×; Carl Zeiss Meditec Dentistry, Oberkochen, Germany). Diagnosis was also performed using percussion, transillumination, thermal and electric pulp testing, and periapical radiographs. Information was given to these patients concerning the possibility of maintaining the teeth with a treatment plan consisting of root canal treatment and coronal coverage; however, after being advised of the prognosis, 11 of these patients opted for extraction. One of the molars with a clinical diagnosis of reversible pulpitis had to be extracted to accomplish a prosthetic treatment plan.

The remaining 8 of 20 teeth showed severe attrition leading to enamel loss and dentin exposure (4 mandibular incisors, 2 mandibular canines, and 2 mandibular premolars). None of these teeth showed clinical evidence of cracks. Three of these teeth were extracted because the patients presented with severe pain and did not accept any treatment aimed at retaining these teeth; the other 5 teeth were asymptomatic and were extracted to accommodate a prosthetic treatment plan (full denture preparation). All patients gave consent for histologic examination of their teeth.

Histologic Processing

Immediately after extraction, the following measures were undertaken to allow proper fixation of the pulp tissue and a correct orientation of the specimen within the paraffin block. To obtain histologic sections perpendicular to the crack line(s), the roots were separated 2–3 mm apically to the root canal orifices, and the crowns were ground under magnification with high-speed diamond burs under water spray on a mesiodistal, buccolingual, or axial plane until 1 or 2 pulp horns were encountered. Subsequently, specimens were immersed in a 10% neutral-buffered formalin solution for at least 48 hours. Demineralization was performed in an aqueous solution consisting of a mixture of 22.5% (v/v) formic acid and 10% (w/v) sodium citrate for 3 to 4 weeks with the end point being determined radiographically. All specimens were then washed in running tap water for 48 hours, dehydrated in ascending grades of ethanol, cleared in xylene, infiltrated, and embedded in paraffin (melting point 56°C) according to standard procedures. With the microtome set at 4–5 μm, meticulous longitudinal serial sections were taken until the entire pulp chamber volume was exhausted. Approximately 500 to 1000 sections were cut for each molar tooth. Four to 6 sections were collected on each glass slide. Every fifth slide was stained with hematoxylin-eosin for screening purposes. Once the slides with the sections encompassing the crack line and the pulp space were identified, they were all stained with either hematoxylin-eosin for assessment of inflammation or a modified Brown and Brenn stain for the visualization of bacteria (21, 22). Coverslips were then placed on the slides, and the sections were examined under a light

TABLE 1. Case Description

Case#	Patient	Sex	Age	Tooth	Symptoms	Crown restoration	Clinical diagnosis	Pulp diagnosis	Histologic diagnosis
1	SC	F	61	31	Yes	No	Crack	Irreversible pulpitis	Irreversible pulpitis
2	GG	M	31	19	Yes	Deep class I amalgam	Crack	Irreversible pulpitis	Irreversible pulpitis
3	CF	F	28	31	Yes	Medium class II amalgam	Crack	Irreversible pulpitis	Irreversible pulpitis
4	GM	M	52	5	Yes	No	Crack	Irreversible pulpitis	Irreversible pulpitis
5	LR	F	56	30	Yes	Minimal class I amalgam	Crack	Irreversible pulpitis	Irreversible pulpitis
6	TF	M	54	14	Yes	Medium class II amalgam	Crack	Pulp necrosis with AP	Partial pulp necrosis
7	CE	F	24	18	Yes	Deep class I amalgam	Crack	Irreversible pulpitis	Reversible pulpitis
8	SMT	F	35	14	Yes	Deep class II amalgam	Crack	Irreversible pulpitis	Irreversible pulpitis
9	FM	F	77	17	Yes	Deep class I amalgam	Crack	Pulp necrosis with AP	Pulp necrosis
10	RC	F	42	5	Yes	Deep class II amalgam	Crack	Irreversible pulpitis	Irreversible pulpitis
11	OM	F	43	19	No	Shallow occlusal and cervical composites	Craze	Reversible pulpitis	Reversible pulpitis
12	ML	F	65	30	Yes	No	Endo-perio lesion	Irreversible pulpitis	Irreversible pulpitis
13	PS	F	61	24	No	No	Attrition	Vital pulp	Atrophic pulp
14	PS	F	61	23	No	No	Attrition	Vital pulp	Atrophic pulp
15	OC	M	75	22	No	No	Attrition	Vital pulp	Atrophic pulp
16	OG	M	69	21	Yes	No	Attrition	Irreversible pulpitis	Irreversible pulpitis
17	MJ	F	62	23	Yes	No	Attrition	Pulp necrosis	Pulp necrosis
18	SV	M	63	20	Yes	No	Attrition	Pulp necrosis	Pulp necrosis
19	PA	F	72	26	No	No	Attrition	Vital pulp	Atrophic pulp
20	PA	F	72	27	No	No	Attrition	Vital pulp	Atrophic pulp

F, female; M, male.