

# Light and SEM observation of internal root resorption of a traumatized permanent central incisor

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## Summary

A clinical case report is presented which illustrates internal root resorption of a traumatized tooth. Light and scanning electron microscope were used to further examine the defect. An explanation of the morphological structure of the resorption is discussed.

**Key words:** internal resorption, trauma

## Introduction

Internal root resorption following dental injury is a rare finding in the permanent dentition (Andreasen 1970, Crona-Iarsson *et al.* 1991). This pathological process, which is often free of clinical symptoms, begins within the pulp canal space and progresses toward the external surface of the root. It is usually discovered during routine radiographic examination as a well-defined, enlarged, radiolucent area extending from the pulp space. The exact aetiology of internal root resorption is still unknown; however, traumatic tissue injury and infection have been suggested (Schroder & Granath 1971, Andreasen 1981).

Two types of internal root resorption; internal replacement resorption and internal inflammatory resorption, have been described by Andreasen (1981). The internal replacement resorption is characterized by metaplasia of the normal pulp tissue into cancellous bone-like tissue. The internal inflammatory resorption is characterized by the transformation of normal pulp tissue into granulation tissue.

Dentinoclasts, the cells that resorb dentine, are multinucleated cells with intense tartrate-resistant acid phosphatase activity similar to osteoclasts (Nilsen &

Magusson 1981, Wedenberg & Zetterqvist 1987). These enzyme properties allow the dentinoclasts to produce a large resorption pit or lacuna on the hard tissue surface (Pierce 1989). Histological examination of the extracted tooth could be used to confirm the diagnosis of internal root resorption. Thus, the purpose of this article was to use light and scanning electron microscopy to examine a traumatized tooth with evidence of a perforating quiescent internal resorption.

## Case report

A 17-year-old female with a history of trauma 5 years previously, reported to the primary care clinic of King Saud University to restore a cavity on the cervical area of the buccal surface of the maxillary left central incisor. The patient had noticed the cavity for a week. She reported no pain or swelling and the medical history was non-contributory. The clinical examination revealed a crown fracture of both upper central incisors involving enamel and dentine. The teeth had not been restored since the trauma. Electric sensitivity testing using an Analytic Technology Pulp Tester (Redmond, WA, USA) as well as a cold test on both upper central incisors were non-responsive. The left central incisor showed slight tenderness to palpation. A 4-mm periodontal pocket could be probed on the buccal aspect of the tooth with otherwise good oral hygiene. Periapical radiographic examination revealed a large radiolucent lesion in the coronal and cervical areas of the root canal of the left central incisor (Fig. 1). A periapical radiolucency was noticed. The tooth was considered unrestorable, and was extracted and prepared for light and scanning electron microscopic (SEM) examination.

## Light microscopy preparation

The extracted tooth was split longitudinally through the buccal lesion and fixed in 10% buffered formalin. One-

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Fig. 1. Periapical radiograph of maxillary central incisors. Both teeth have crown fracture involving the enamel and dentine. Internal resorption is evident in the mid-coronal and cervical areas of the left central incisor.

half of the tooth was decalcified for 1 day in 5% nitric acid at room temperature. The nitric acid solution was changed daily and agitated by hand. After decalcification, the specimen was rinsed in running tap water for 4 h, dehydrated in alcohol and embedded in paraffin. Serial sections (5  $\mu$ m) were cut longitudinally and every other slide was stained with haematoxylin and eosin. Selected sections were stained with the Brown and Brenn technique for the demonstration of bacteria.

#### *SUM preparation*

The remaining half of the tooth was dehydrated in a graded series of alcohol and carbon dioxide critical point-dried, mounted on an aluminium stub and gold coated with the sputter technique. A Jeol ISM-T330A (Tokyo, Japan) scanning electron microscope at 10 kV was used to examine the specimen.

#### *Observation*

Examination under light microscopy revealed an external root resorption located at the mid-root area away from the resorption cavity associated with the internal root resorption. A periapical granuloma remained attached to the root apex. The internal root

resorption was evident, the pulp space was empty and only few remnants of necrotic tissue were observed. Dentinoclasts were not seen in those sections stained with Brown and Brenn stain but bacteria were observed in the dentinal tubules, mainly in the coronal pulp space (Fig. 2),

The SUM examination showed numerous odontoblast process-like structures with smooth surfaces emerging from the apical part of the resorptive cavity (Figs 3 and 4). The dentine wall was extensively resorbed and difficult to identify. Bacteria and filaments were observed in complex arrangements (Fig. 5).

#### **Discussion**

After histological evaluation, it appeared that this tooth had initially undergone pulp inflammation followed by



Fig. 2. Bacteria in the dentinal tubules of the coronal pulp space. Section stained according to the Brown and Brenn technique (original magnification  $\times 1001$ ).

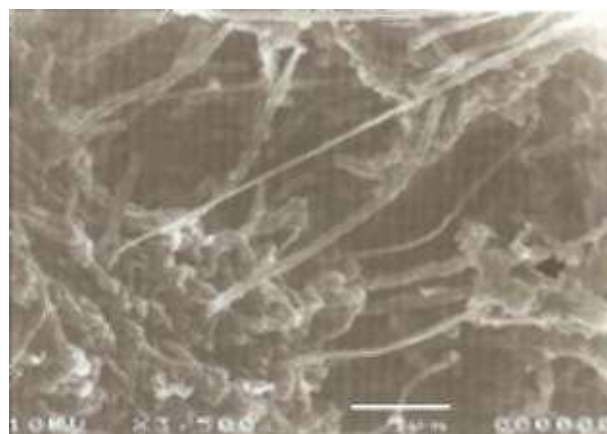


Fig. 3. Scanning electron micrograph showing number of sheet-like structures that could be interpreted as odontoblast process-like. Note the resorbed dentine (arrow). Bar = 5  $\mu$ m.

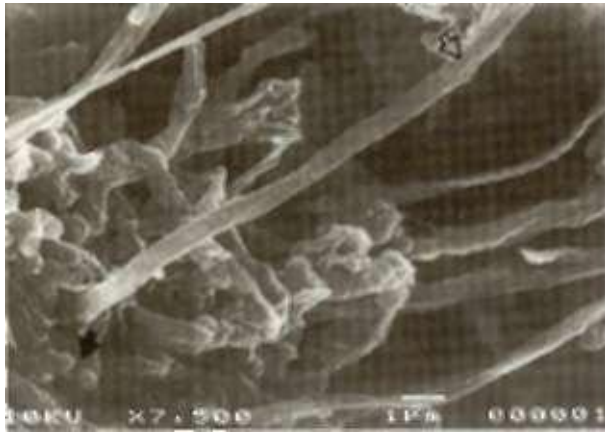


Fig. 4. High magnification of Fig. J showing depressions on the trunk of the process (open arrow). Um- numbers of bacteria (cocci) can be seen (arrow). Bar = 1  $\mu$ m

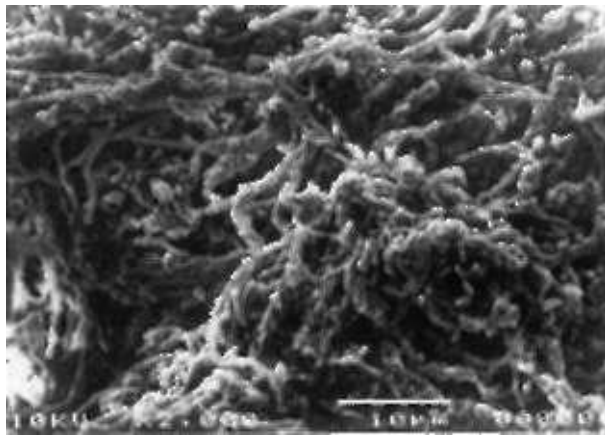


Fig. 5. Scanning electron micrograph showing large number of entangled bacterial threads-like structure covering the 100th structure near the cavity (buccal) opening. Bar = 10  $\mu$ m.

bacterial infection and necrosis. During this progression of pulpal inflammation, pulp metaplasia with massive internal dentine resorption occurred. This pulpal inflammation, necrosis and infection led to subsequent periapical bone loss. At the time of this examination the pulp was necrotic.

In this investigation, as in others (Kelley *et al.* 198 J. Szabo *et al.* 1984), the carbon dioxide critical-point drying technique for SEM analysis was used. This served well and preserved the tissue with little adverse effects, such as shrinkage, as water is rapidly removed from the tissues.

Wednberg & Zetterqvist (1987) observed a large number of inflammatory cells, bacteria and dentinoclast-like cells associated with internal resorption. Bacteria were only histologically detectable in teeth with the most rapidly progressing resorption. The observed

bacteria in the case described here could be related to the trauma resulting in crown fracture with exposed dentine. A report of late management of enamel and dentine fracture by Al-Nazhan *et al.* (in press) showed 53% pulp necrosis. This demonstrates that leaving traumatized dentine uncovered for a longer period of time may expose it to saliva and other types of bacterial contamination. This may result subsequently in bacterial invasion through the open dentinal tubules leading to pulp necrosis (Otgart *et al.* 1974).

The recent buccal cavity which finally brought the patient for treatment could be the result of the expanding resorption process that eventually eroded the surface of the root.

In the examination of this case, sheet-like structures at the site of resorption were observed. In Brown and Brenn stain and the SEM it appeared that these structures could be bacterial filaments or rod-shaped organisms. These were located in the apical areas of the resorption close to the cavity wall of the pulpal side. Similar morphological structures have been reported by Molven *et al.* (1991) and El-Labban *et al.* (1991). Such bacteria were not seen at the site of resorption.

Ohgushi & Fusayama (1975) and Yamada *et al.* (1983), using electron microscopy, reported that the odontoblast processes could extend through the inner areas of carious dentine. In this study, the odontoblast processes remained, in some areas, with well-preserved structure. In addition, the depression that appeared in the surfaces of the odontoblastic process reported by Yamada *et al.* (1983), was observed. These processes were clearly different from the morphology of the bacterial filaments (Fig. 4). According to Thomas & Carella (1983), the sheet-like structures of vital odontoblasts are extracellular and correspond to the lamina limitans. The morphological differences between the odontoblast process, the lamina limitans and the entangled bacterial filaments, together with observations by Ohgushi & Fusayama (1975) and Yamada *et al.* (1983) suggest that the sheet-like structure in this reported case could be remaining mummified odontoblastic processes.

Using cytochemical detection of acid phosphate activity in combination with morphological techniques. Wednberg & Undskog (1985) reported that infected teeth, observed for up to 10 weeks, had more extensive and lasting bacterial colonization in the dentine wall compared with non-infected. This was related to the activity of the macrophage-like resorbing cells engaged in the resorption of mineralized tissue. They also observed that the spreading of the resorbing cells occurred more readily when bacteria were not present.

In this case, the resorbing cells were not seen when bacteria were observed at the site of resorption, probably because the pulp was necrotic.

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