Healing of root resorption: a case report

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External resorption is sequelae of necrotic periodontal membrane over a large area of root following an injury to the tooth. This usually occurs after severe dental injuries such as intrusion, severe luxations or exarticulation injuries complicated by a prolonged extra oral period. This case report presents a clinical and radiographic follow up (13 months) of treatment of inflammatory external root resorption on maxillary central incisor using Vitapex®. Gradual healing of resorption was observed radiographically with no tenderness or pathological mobility.
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INTRODUCTION

Unlike bone, which undergoes resorption and apposition as a part of continual remodelling process, the roots of the permanent teeth are not normally resorbed unless as a common sequelae after reimplantation of avulsed tooth.[1]

Osteoclast and osteocytes are the main cells involved in the resorption of hard tissue, while the former resorbs both bone and other hard tissue; the latter resorbs only the bone.[1]

The stimuli for resorption are enzymes, oxygen tension, hormones, locally produced chemical mediators and electric current.[2]

The root being resistant to resorption both on the external surface and the pulpal aspect internally, does not respond to these stimuli.

The connective tissue of periodontal ligament, the outer most layer (cementoblasts and cementoid) and the innermost intermediate layer of cementum have been thought to play some role in the resistance of external surface of root resorption, while the predentin and odontoblasts are accepted as the resorption inhibitors internally. On the most external aspect of the cementum is a layer of cementoblast covering a zone of non-mineralized cernentoid. This cementoid layer is predominantly organic in nature as is the predentin layer internally. These two are thought to be the key in resisting resorption.

Another hypothesis is that elastic cells are attracted and attached only to mineralized tissue.[3] Thus, an injury that results in removal of the precernentum or predentin or any process that results in mineralization of the organic matrix predisposes the root to resorption.

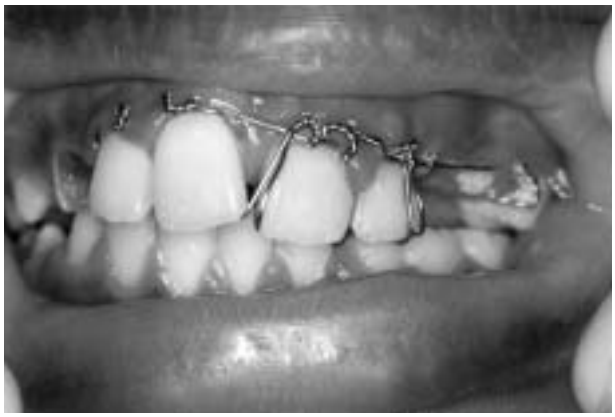

Figure 1.

CASE REPORT

A 13-year-old patient visited the Department of Pedodontics, Yenepoya Dental College, with a complaint of pain and mobility of the front tooth. History revealed that the patient had a fall about a month ago and his left maxillary central incisors was avulsed along with severe displacement of left maxillary lateral incisors.

He had visited a private practitioner, who replanted the avulsed tooth and stabilized with wires. On examination there was improper wiring on the anterior teeth without proper stability in relation to 21 and 22, (Figure 1).

Twenty-one (21) was mobile and tender on percussion with marked gingival inflammation. The root canals of both 21 and 22 were open suggesting extirpation of the pulp.

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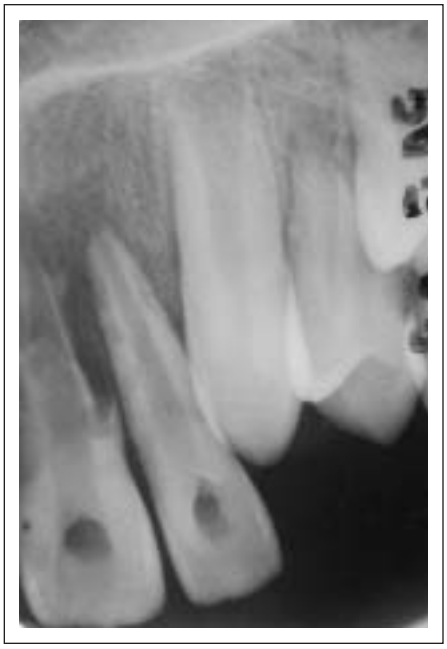

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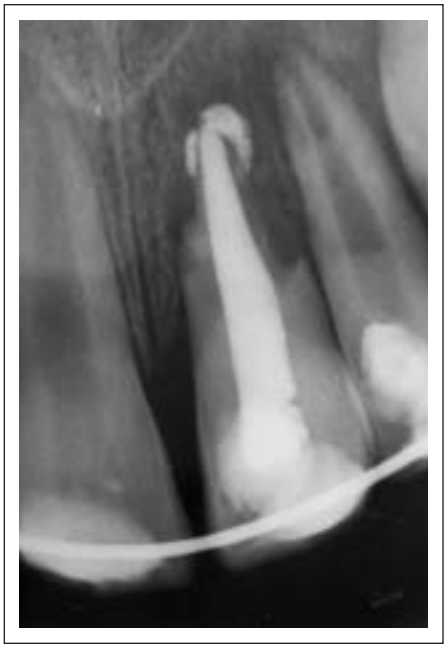

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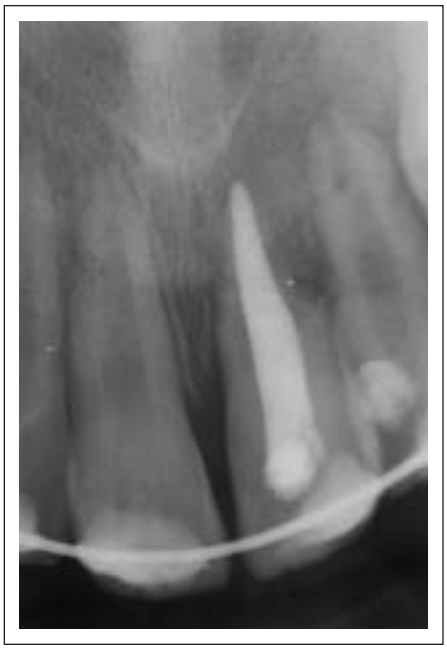

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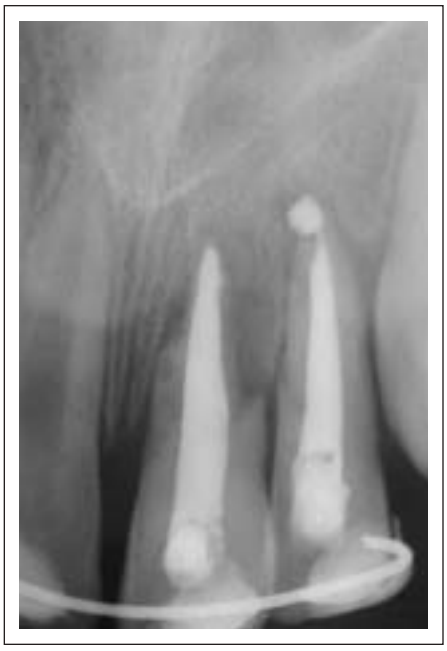
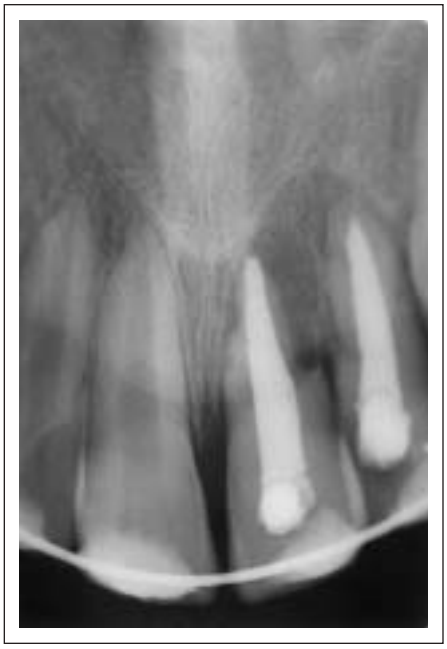


Figure 5.

Radiographic examination revealed areas of radiolucency along the apical and lateral surface of the root and surrounding bones (moth eaten appearance) with loss of lamina dura suggesting external inflammatory root resorption. Twenty-two (22) showed apical radiolucency (Figure 2). Since the treatment of inflammatory resorption is directed towards preventing or removing the stimulus for inflammation, the old wire splinting was removed and a new semi-rigid acid etch composite splinting was done (as the tooth was mobile) followed by debridement of the root canals of 21. Then the canal was obturated with Vitapex [Ca $(OH)_2$ and iodoform].

Immediate postoperative radiograph showed extrusion of excess material in the periapical region, (Figure 3) which resorbed after 2 weeks (Figure 4). The same was observed in 22 (Figures 5, 6). Recall examination after 2 weeks showed that the tooth was asymptomatic with no mobility or tenderness on percussion. The splint was



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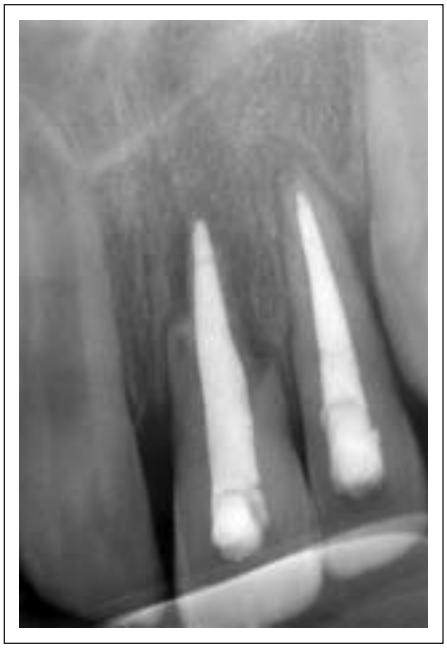

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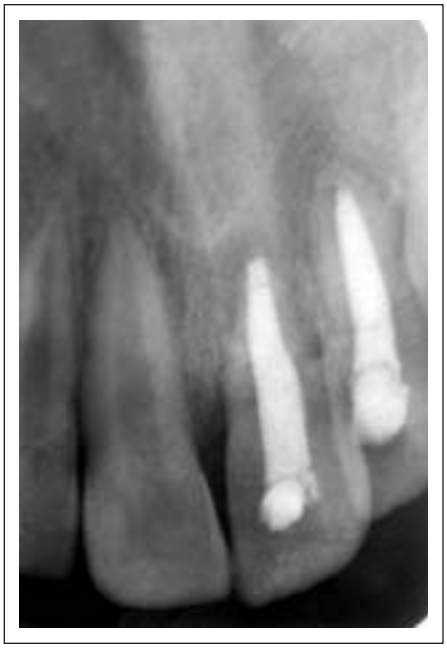

Figure 8.

removed after 2 weeks and the patient was instructed not to bite on his front teeth.

In a 12-month follow up, series of radiographs showed healing of inflammatory resorption and new bone was deposited (Figure 7). At the end of 13 months the external resorption had ceased completely (Figure 8) with no mobility or tenderness clinically. A conventional gutta percha obturation was planned in future.

DISCUSSION

Minor injuries to the periodonta1 ligament and/or cementum due to trauma or contamination with bacteria induce small resorption cavities on the root surface. If these resorption cavities expose dentinal tubules and the root canal contains infected necrotic tissue, toxins from these areas will penetrate along the dentinal tubules to the lateral periodontal tissues and provoke an inflammatory response.

This will intensify the resorption process, which advances towards the root canal. Inflammatory resorption is especially frequent and aggressive after replantation in young patient. This is probably due to the wide dentinal tubules and a thin protective cementum cover.

Andersen[6] has shown that resorption defects occur on the root surface adjacent to the areas of damage to the periodontal ligament during avulsion or extended drying before replantation.

According to Andersen,[6] inflammatory resorption is dependent on the following conditions:

1. Injury to the periodontal ligament.
2. Exposure of dentinal tubules.
3. Communication of the exposed tubules with necrotic pulp as with a leucocytic zone with bacteria.
4. The presence of maturation factors.

These conditions contribute in the present case. The improper splinting, which caused gingival inflammation,

(Figure 1) could have prevented the inflammatory response from resolving in the marginal gingiva and possibly around the replant.

Inflammatory resorption is the mechanism of eliminating infected calcified tissue from the body; osteoclasts acting as specialized macrophages actively participate in the healing process to repair traumatized tooth and bone.[6]

Endodontic treatment effectively prevents inflammatory resorption, if the necrotic pulp is removed before the initiation of bacterial infection. Inflammatory resorption is difficult to eliminate if the endodontic treatment is delayed more than 3 weeks following replantation.[6]

Andersen thus emphasizes the importance of early endodontic treatment of avulsed teeth, especially those with a compromised or necrotic periodontal ligament. If the resorption process is allowed to progress and involve large areas of root surface replacement resorption (ankylosis) can take over once inflammatory resorption has been arrested by endodontic therapy. The inflammatory process removes the necrotic debris from the root surface. If the infection is not present to sustain the inflammation, healing will follow by a competition between the remaining vital periodontal cells and in growth of alveolar bone across the periodontal ligament space.[4] If less than 20% of the root surface is involved, reversal of ankylosis may occur.[13] If a large area is affected, as in this case, it is extremely difficult for the periodontal ligament cells to repopulate the entire area before bone producing cells move across the periodontal ligament space.

$Ca(OH)_2$ is the material of choice in treating inflammatory resorption. It is an effective antibacterial agent and it has been reported to favorably influence the local environment at the resorption site promoting healing.[3] It changes the environment in the dentin to a more alkaline pH which slows the action of resorptive cells and promote hard tissue formation. But it has to be changed every 3 months within the range of 6 to 24 months.

Other materials like Ledermix and Calcitonin is been shown to be an effective medication in the treatment of inflammatory root resorption.

A commercial product named Vitapex (J. Morita) containing a viscous mix of $Ca(OH)_2$ and iodoform in a syringe with disposable tips was used in this case. The main ingredients of Vitapex are iodoform 40.4%, $Ca(OH)_2$ 30.3% and silicone 22.4%.

Vitapex when extended into periapical or furcal area can either diffuse away or be resorbed in part by macrophages in a short time as one or two months. Studies have shown that iodoform paste are bactericidal to microorganism in the root canal and lose only 20% of potency over a 10 years period.[10]

Bone generation has been clinically and histologically documented after using iodoforrn and $Ca(OH)_2$ mixtures.[9] According to Andeasen replacement resorption can take place once inflammatory resorption has been arrested by endodontic therapy.[12] This is more in case of young patients because of high turnover rate of bone forming cells. The same process was noted in the present case. Though there is no treatment for replacement resorption, it is worth an effort to try to slow down the process and maintain the tooth as long as possible in the arch for esthetic, mastication, natural space maintenance and above all psychological uplift of the young minds.

CONCLUSION

Once replacement resorption was developed there is no treatment known that will affect the progress. Therefore prevention of the development of the process is of utmost importance. Since nothing can be done about the extent of the initial injury to the attachment apparatus, the main emphasis is:

1. Maintaining the health of the remaining periodontal ligament cells so that they are available to repopulate the demanded root surface and re-establish a normal periodontal ligament space.
2. Removal of the irreversibly damaged periodontal ligament before replantation, to minimize inflammation afterward.

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