

External inflammatory and replacement resorption of luxated, and avulsed replanted permanent incisors: a review and case presentation

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Abstract – External inflammatory resorption and replacement resorption are complicating factors that may result from traumatic dental injuries when the tooth is luxated or avulsed and replanted. Resorption may, ultimately, result in loss of the tooth. However, with appropriate treatment, the prognosis for these teeth is greatly improved, with the possibility of preventing or arresting resorption. The purpose of this paper is to review these trauma entities, to discuss factors that influence the occurrence of resorption and to describe the most appropriate treatment. A case is presented, illustrating both resorption entities, but with varying outcomes.

In severe luxation and avulsion injuries, where crushing or other damage of the periodontal ligament occurs, complicating sequelae may result in root resorption. Loss of pre-cementum, cementoblasts, and epithelial rests of Mallassez results in the denuded root surface being chemotactic to hard tissue resorbing cells. Macrophages and osteoclasts subsequently remove damaged periodontal ligament and cementum. Further events are determined by:

- Eventual exposure of dentinal tubules
- Content of the pulp, whether ischaemic and sterile or necrotic and infected
- Presence of adjacent vital cementoblasts.

The combination of these factors may lead to external root resorption at the site of injury (1–3).

The development of *inflammatory root resorption* is directly related to damage of the periodontium at the time of trauma, and the presence of bacteria within the root canal and dentinal tubules (4).

The development of *replacement resorption* depends on both the degree of damage to the periodontium at the time of injury, and the extent to which the viability

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of the periodontal ligament cells remaining on the root surface are maintained (4).

Inflammatory resorption

When pulp necrosis occurs following luxation and avulsion injuries, the necrotic tissue is susceptible to bacterial contamination. The combination of bacteria in the root canal and cemental damage on the external root surface results in external inflammatory root resorption (1, 5). If the resorptive process exposes dentine, toxins from bacteria present in the tubules and/or the infected root canal can be transmitted to the periodontal ligament. Stimulators of hard tissue resorption (including macrophage chemotactic factor, osteoclast activating factor, prostaglandins) are released (1). Inflammation in the periodontal ligament and osteoclastic activity lead to resorption of the lamina dura and adjacent bone. The process usually progresses until the root canal is exposed.

External inflammatory root resorption can occur rapidly in young teeth because the dentinal tubules are wide and allow the irritants to move freely to the external surface of the root (5).

External inflammatory and replacement resorption



Fig. 1. External inflammatory root resorption, affecting tooth 11. Note destruction of adjacent alveolar bone.

Diagnosis of inflammatory resorption

Radiographically, external inflammatory resorption is characterised by a progressive loss of tooth substance associated with a persistent or progressive radiolucency in the adjacent alveolar bone (6) (Fig. 1).

Treatment of inflammatory resorption

Elimination of bacteria from the root canal and/or dentinal tubules by endodontic therapy will arrest the resorptive process. An interim root canal dressing of calcium hydroxide is recommended prior to final restoration. The mechanism by which calcium hydroxide participates in the reparative process is unclear (6,7), but success rates of up to 97% have been reported using this method (6).

Once inflammatory resorption has been arrested, the resulting defect is repaired by cementum or bone, according to the type of vital tissue found next to the resorption site (periodontal ligament or bone marrow-derived tissue). Healing may occur irrespective of the extent of resorption and the amount of root substance lost (6). Ankylosis may occur at a site of previously arrested inflammatory resorption. This is believed to occur as a result of the initial periodontal injury, rather than as a result of calcium hydroxide therapy (6).

Replacement resorption

When extensive damage occurs to the innermost layer of the periodontal ligament, competitive healing

events take place. Healing from the socket wall (creating bone via bone marrow-derived cells) and healing from adjacent periodontal ligament (creating cementum and Sharpey's fibres) occurs simultaneously.

If less than 20% of the root surface is involved, a transient ankylosis may occur, which can later be resorbed due to functional stimuli, provided the tooth in the healing period is stabilised with a splint which allows a minimum amount of mobility, or is non-splinted (1, 3). In larger injuries ($>4 \text{ mm}^2$), a permanent ankylosis is created. The tooth thus becomes an integral part of the bone remodelling system, the resorbing cells being, primarily, osteoclasts. Subsequently, osteoblasts replace the resorbed areas of the root with bone (3).

In children, replacement resorption leads to loss of ankylosed teeth usually within 1–5 years. In adults, replacement resorption occurs more slowly, often allowing the tooth to function for many years (1–3).

Diagnosis of replacement resorption

Clinically, the affected tooth is immobile, and exhibits a high percussive tone. Radiographically, the periodontal ligament space is absent, and a direct union is seen between alveolar bone and the root (Figs 2 and 3). In time, infra-occlusion relative to adjacent teeth can be seen both clinically and radiographically.

Treatment of replacement resorption

While appropriate endodontic therapy is effective in the treatment of external inflammatory resorption, replacement resorption cannot be arrested or repaired.



Fig. 2. Arrest of external inflammatory root resorption, tooth 11, following treatment with calcium hydroxide. Note healing of alveolar bone.



Fig. 3. Replacement resorption affecting apex 11, and mid-root fracture of 11. Note that 21 has been root filled, and no ankylosis is evident.

Tooth avulsion

An avulsion injury constitutes a serious assault on the gingiva, the alveolus, the periodontium, the pulp and the cementum, and may cause damage to the dentine of the root (8). It has been shown that in animals, under ideal conditions, complete healing of the dental and supporting tissues can occur following replantation of an avulsed tooth (9). Such ideal conditions rarely occur, however, in humans and a wide range of outcomes can result. Root resorption is common, with a reported prevalence of 57–80% (8, 10–13).

The medium in which the tooth has been stored affects the levels of root resorption and pulp healing. Prolonged drying of the root is detrimental due to loss of vitality of the periodontal ligament and dehydration of the pulp (10, 14, 15).

Some studies have found that extended extra-alveolar time is a good predictor of resorption (11, 13, 16), while others have not (17). All these studies, however, have shown that those teeth that were kept dry eventually developed root resorption. Total extra-alveolar time has less effect on the outcome provided the tooth has been stored in a wet medium (8, 17).

Donaldson & Kinirons (13) found that the risk of early resorption is increased in teeth that have additional damage or contamination of the root, or are kept in dry conditions for longer than 15 min. They found that dry time is the most crucial clinical factor

associated with the development of post-replantation root resorption. A previous study by Kinirons et al. (8) indicated that the risk of resorption increases dramatically after 5 min of dryness, with the probability of resorption increasing by 29% for every additional 10 min of dryness.

If the periodontal ligament left attached to the root surface does not dry out, the consequences of tooth avulsion are usually minimal. The hydrated periodontal ligament cells will maintain their viability, allowing them to reattach on replantation without causing any more than minimal destructive inflammation. However, if excessive drying occurs before replantation, the damaged periodontal ligament cells will elicit a severe inflammatory response over a diffuse area on the root surface (5).

The consequences of tooth avulsion appear directly related to the severity and surface area of the inflammation on the root surface, and the resultant damaged root surface that must be repaired. Treatment strategies aim to limit the extent of the peri-radicular inflammation, thereby tipping the balance towards favourable (cemental) rather than unfavourable (inflammatory resorption or replacement resorption) healing (5).

Treatment of avulsion injury

Treatment is aimed at limiting damage to the periodontal attachment apparatus and the resultant inflammation, and preventing or arresting inflammatory resorption. Minimising the extra-oral dry time is the most important factor (13). When this factor cannot be adequately controlled, and the tooth has been dry for more than 15 min (13), steps can be taken to slow the inevitable replacement resorption (5). Placing the tooth in a suitable storage medium can prolong the viability of the periodontal ligament. Suitable media, in order of preference, are: Hank's pH balanced salt solution (Gibco™, Invitrogen Corporation, 1600 Faraday Ave., Carlsbad CA 92008, USA), Via Span® (Belzer UW-CSS, DuPont Pharmaceuticals, Wilmington, DE, USA), cold fresh milk, saliva, physiologic saline (5, 18).

Periodontal ligament cells can be expected to survive a dry time of 15 min or less (13), but are unlikely to survive a dry time of greater than 60 min (5). Teeth that have been dry for greater than 15 min, but less than 60 min pose a treatment problem. It is probable that the root surface will contain some viable periodontal cells, and some that will act as stimulators of inflammation. It has been suggested that the use of enamel matrix derivative Emdogain® (Biora, Malmo, Sweden) may encourage periodontal healing in such replanted teeth (5).

When a tooth has had an extra-oral dry time of greater than 60 min, the periodontal ligament is not

expected to survive (5). Pre-treatment of such a tooth, prior to its replanting, will render it more resistant to resorption. Traditionally, stannous fluoride has been used for the purpose (5, 18), but alternative therapies have recently been described which appear to offer more encouraging results (19–21). Further research is continuing to determine the optimal treatment protocol for such teeth.

Systemic antibiotics given at the time of replantation are recommended to prevent bacterial invasion of the necrotic pulp, thereby avoiding inflammatory resorption (5, 18). Both inflammatory resorption and replacement resorption may be diagnosed within 2 months of replantation of an avulsed tooth but, frequently, are not detected until after 6 months. If resorption is not detected within 2 years, the risk of resorption is considerably reduced (9, 15).

Pulp therapy

Several authors have advocated early removal of the pulp of replanted teeth in order to minimise or prevent inflammatory resorption (22, 23). However, Andreassen (9) recommends delaying endodontic treatment for 1 week after replantation to prevent the development of ankylosis and inflammatory resorption, and allow time for reattachment of periodontal fibres. If pulp therapy of a replanted tooth with a closed apex is initiated within 7–10 days of injury, the pulp should be necrotic with little or no infection (5, 24).

For teeth with a wide open apex, however, endodontic treatment may be delayed, as revascularisation of the pulp is possible (9). Studies in animals have demonstrated enhanced revascularisation when the tooth has been soaked for 5 min in doxycycline prior to replantation (25, 26). A recent paper (5) citing these studies recommends the use of topical doxycycline prior to the replantation of a tooth with an open apex. Kinirons et al. suggest that pulp treatment should be delayed in those teeth with wide open apices that have been replanted promptly (15). They note that all other replanted teeth should have the pulp removed as soon as the tooth is stable enough, ideally within 10 days of trauma (18), and they caution that where the clinician has chosen to delay endodontic treatment, the patient should be carefully reviewed so that the pulp can be removed at the first sign of inflammatory resorption.

Kinirons et al. found that the incidence of inflammatory resorption was increased when pulp extirpation was delayed more than 20 days (15). The use of an anti-inflammatory agent containing antibiotic and steroid (Ledermix[®], Haupt Pharma Wolfratshausen GmbH, Pfaffenrieder Strasse 5, 82515 Wolfratshausen, Germany), placed in the root canal prior to placement of calcium hydroxide, has recently been studied by Trope (5). The author claims that such use of Ledermix[®]

in replanted teeth leads to more favourable healing by suppressing the inflammatory response.

Splinting

A splinting technique that allows physiologic movement of the tooth during healing, and that is in place for a minimal time period results in a decreased incidence of ankylosis (5, 9, 27–30). Kinirons et al. demonstrated that the best outcome was achieved if the period of splinting was 10 days or less (15).

Case presentation

A case is presented of a 10-year-old boy who sustained trauma to his maxillary central incisors in a fall from his bicycle. Upper right central incisor tooth 11 suffered extrusive luxation, while upper left central incisor tooth 21 was avulsed.

The avulsed tooth was placed in milk within 5 min of avulsion, and was replanted by a dentist within 45 min. The same operator also repositioned the luxated tooth, 45 min after trauma. A 5-day course of Amoxycillin was prescribed, and the teeth were splinted for 5 days with a non-rigid splint. Twelve days after avulsion, the pulp of the upper left central incisor was extirpated. Polyantibiotic paste¹ was placed in the root canal, and this was replaced with non-setting calcium hydroxide after 8 days.

Endodontic therapy of the upper right central incisor was planned. Unfortunately, the patient failed to attend scheduled appointments. The patient next attended, unscheduled, 6 months later. Examination revealed a labial sinus associated with the upper right central incisor, with radiographic evidence of external inflammatory root resorption (Fig. 1). The infected pulp was extirpated from the tooth, and polyantibiotic paste was placed. Two weeks later, this was replaced with non-setting calcium hydroxide. Radiographic evidence of healing of the bony lesion adjacent to the tooth was obtained after 3 months (Fig. 2).

As the prognosis for the right central incisor was considered hopeless, treatment was aimed at arresting inflammatory resorption, repairing and maintaining alveolar bone with a view to facilitating later placement of a prosthesis and maintaining the tooth for as long as possible, with replacement of calcium hydroxide dressings as necessary. A gutta percha root filling was provided for the upper left central incisor.

¹Polyantibiotic paste:

Neomycin sulphate	10.0 g
Polymyxin B sulphate	3.0 m Units
Bacitracin	2.11 g
Nystatin	2.5 m Units
Polyethylene glycol 1300	25.6 g
Polyethylene glycol 1500	11.0 g

Twenty-eight months after the initial trauma, the patient reported another injury to the upper right central incisor. Radiographic examination revealed replacement resorption affecting the apical portion of the tooth, together with a mid-root fracture (Fig 2). The coronal fragment of I1 was extracted. No attempt was made to extract the apical portion of the tooth, as it was anticipated that progressive resorption would eventually result in its replacement by bone. An acrylic partial denture, replacing the lost tooth, was provided. This will eventually be replaced with a permanent restoration, such as a bridge or implant-supported prosthesis. The treatment provided, which arrested inflammatory resorption of the right central incisor and resulted in healing of alveolar bone, will facilitate later placement of an implant, should this be deemed appropriate for the patient.

Discussion

Root resorption following extrusive luxation is rare (the incidence of inflammatory resorption being approximately 5%) (3, 31), particularly when the tooth is repositioned within 90 min (32). In avulsed replanted teeth, however, root resorption is common, with a reported prevalence of 57–80% (8, 10–13).

It is interesting that the tooth that has suffered a more severe injury (21) has responded better to treatment with, to date, no evidence of resorption. This illustrates how prompt, appropriate treatment can help to prevent resorption and its disastrous consequences.

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