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CASE REPORT

Rapid internal root resorption of the permanent incisor teeth after trauma: a case report

Kazuhito Yoshikawa, Jun Sato, Emi Yamashita, Noritaka Ohga, Takuya Asaka and Yoshimasa Kitagawa

ABSTRACT: Internal root resorption of permanent teeth is a rare condition. Although its etiology and pathogenesis are not fully understood, trauma has been considered a contributing factor. The treatment and prognosis of internal root resorption of permanent teeth is very challenging. In this report, we describe a patient who experienced internal root resorption of the permanent incisors soon after a traumatic episode. This suggests conservation of permanent teeth may be enhanced by early detection and treatment of this condition.

Key words: internal root resorption, permanent teeth, trauma, rapid occurrence

Introduction

Internal root resorption of the permanent teeth is rare and usually asymptomatic, with prevalence rates ranging from 0.01% to 0.2%. Although its etiology and pathogenesis are only partially understood, trauma and persistent chronic pulpitis, as well as orthodontic treatment, may be contributing factors. Because of the rarity of internal root resorption, there have been few studies regarding its time of onset after trauma. This case report demonstrates the importance of frequent and close follow-up evaluation of the affected teeth after traumatic injury. And we found here that immediate root canal treatment may stop the progression of internal root resorption.

Case report

A 13-year-old boy presented with pain in a maxillary incisor one hour after being kicked in the maxillofacial region by a classmate. He had no medical problems and he was conscious. He experienced perforating wound on the right upper lip and dislocation of the right central and lateral maxillary incisors to the palatal side (Fig. 1A-C). The gingiva around the incisors was injured and bleeding (Fig. 1C). The left central incisor showed a crown fracture with pulp exposure (Fig. 1C). There were no abnormalities in occlusion of the bilateral molar teeth. His range of mouth opening, 45mm, was normal. Panoramic radiography revealed no evidence of fracture of the maxilla or mandible (Fig. 2A). Dental radiography indicated dislocation of the right maxillary central and lateral incisors to the palatal side, along with partial fracture of the crown of the left maxillary central incisor.

Fig. 1 Clinical findings at the first visit, showing (A) piercing lacerations on the right upper lip, along with some bleeding; (B) perforation between the intra- and extraoral wounds; and (C) dislocation of the right maxillary central and lateral incisors to the palatal side, along with partial fracture of the crown of the left maxillary central incisor.
was clinically diagnosed with lacerations on the right upper lip, palatal luxation of the right central and lateral maxillary incisors, and chipping of a partial crown of the left maxillary central incisor with exposure of the pulp. Following written informed consent from the patient and his parents, he underwent an emergency operation under local anesthesia. The wound on his lip was sutured, and the dislocated teeth were reduced along the alveolar process by hand into their normal position. The teeth were subsequently fixed with wire, 0.5mm in diameter, and adhesive resin (Super Bond®; Sun Medical Co., Ltd., Shiga, Japan). Finally, pulpectomy was performed on the left central maxillary incisor. Dental radiography immediately after the operation revealed that the reduced teeth were in their normal positions, with no abnormalities in the shape of the roots of these three teeth along the alveolar process.

The patient was followed-up every one to two weeks, with the fixed wire removed two weeks after surgery. The recovery of the lacerations of the right upper lip and three treated teeth was clinically uneventful. Two months after trauma, however, dental radiography revealed round and oval lesions in the middle of the root canal in both the right central and lateral maxillary incisors (Fig. 3A). The outlines of the roots were normal, and there were no clinical signs or symptoms, including pain, swelling, or changes in teeth color. Electric vitality tests revealed negative responses for both of these teeth. The patient was diagnosed with internal root resorption of the teeth after trauma and underwent immediate root canal preparations under local anesthesia. When the instrument reached the pulp spaces, continuous bleeding was observed, which was controlled by irrigation with 10% sodium hypochlorite (Neo Cleaner®; Neo Dental Chemical Products Co., Ltd., Tokyo, Japan). The pulps were removed with instrumentation and chemical irrigation with 10% sodium hypochlorite (Neo Cleaner®; Neo Dental Chemical Products Co., Ltd., Tokyo, Japan) and 3% hydrogen peroxide (Oxydol®; Kenei Pharmaceutical Co., Ltd., Osaka, Japan), and the root canals were temporarily filled with calcium hydroxide (Calciplax Plain II®; Nippon Shika Yakuhin Co., Ltd., Yamaguchi, Japan). During treatment, there were no perforations in the lateral walls of the root canals. Three months after trauma, the distal line of the root of the right lateral maxillary incisor became irregular, but the lesions of internal resorption remained stable (Fig. 4). Two years after trauma, the wound in the upper lip had recovered with minor scarring, and there were no remaining clinical symptoms in the three traumatized teeth (Fig. 5A and B). The calcium hydroxide in the root canals was replaced regularly. Dental radiography revealed arrest or minor improvement of the internal root resorption of these two teeth (Fig. 5C). Final filling of the root canal will be performed in the near future.
Fig. 5 Clinical and radiographic findings two years after surgery
(A) Photographs showing that the wound on the upper lip had recovered with minor scarring. (B) No clinical problems were observed in the three traumatized teeth, including changes in crown color, swelling of the gingiva, position of the traumatized teeth, or esthetic disorders. (C) Dental radiograph showing cessation or minor improvement of internal root resorption of the two teeth.

Discussion

Although the etiology of internal root resorption is not fully understood, trauma and persistent chronic pulpitis, as well as orthodontic treatment, have been considered contributing factors. The types of trauma that trigger root resorption, however, have not determined. In 1935, Thoma reported that internal root resorption was found in only 1 of 1000 permanent teeth (0.1 %). Although a recent retrospective study of 889 injured permanent teeth found internal root resorption in 17 of 47 (36 %) root fractured teeth, the details were not described. In the patient described here, we could not detect fracture during the follow-up period.

Due to the rarity of internal root resorption, there have been few studies regarding its time of onset after trauma. One patient was reported to experience internal root resorption after orthodontic treatment for two years, and another experienced internal root resorption of the lower incisors eight months after trauma. The authors described that such rapid progression of internal resorption affected by trauma and involvement of adjacent teeth had not been described previously. In our patient, internal root resorption was detected only two months after trauma. To our knowledge, no other patient has shown such a short interval from trauma to internal root resorption. We found that immediate root canal treatment could stop the progression of internal root resorption. Although we cannot explain the rapidity of internal root resorption, pathophysiology suggests that internal root resorption occurs after odontoblasts necrosis and is associated with chronic partial pulp inflammation and partial pulp necrosis. The progress of internal root resorption is thought to depend on two factors: the pulp tissue in the area of resorption must be vital, and the pulp coronal to the resorption must be partially or completely necrotic, allowing bacterial infection. Our patient’s incisors were dislocated to the palatal side without complete dislocation, and damage to the tissues including the gingival tissues, was not severe. Had the damage to the periodontal tissues, including the periodontal ligament, alveolar bone, and cementum of the root surface, been more severe, the teeth may have experienced progressive external rather than internal root resorption. Moreover, vitality tests on these teeth were negative and pink spots were not observed on the crown, characteristics often accompanying internal root resorption. These observations suggested that the results of vitality tests of teeth, especially after trauma, are not conclusive. Diagnosis of pulp necrosis is quite difficult within several weeks after injury, with negative reactions later becoming positive in some patients.

Histologically, multinucleated giant cells originating from pulp granulation tissue can be found adjacent to the inner root canal wall in teeth experiencing internal root resorption. The active resorption phase is caused by inflammation of granulation tissue. The ability to form granulation tissue is higher in teenagers than in older patients.

It is difficult to determine the timing of final root canal filling to the teeth with internal resorption, because of bleeding and continuous progression of the resorption. In our case, we were not able to perform early final root canal filling because of some bleeding from the root canal.

This case report demonstrated the importance of frequent and close follow-up evaluation of the affected teeth after traumatic injury, especially one to two months after the trauma, even in the absence of clinical symptoms. Early detection of internal root resorption is necessary to conserve the teeth.

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Conflict of interest

The authors declare no conflict of interest.
References