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Management of Acute Apical Abscess Presenting with Rapid Extrusion of a Tooth: A Case Report

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An acute apical abscess (AAA) is a rapid-onset inflammatory condition characterized by spontaneous pain, pus formation, and swelling, often resulting from pulp necrosis. Complications may include systemic manifestations and severe outcomes, such as tooth extrusion. This case report describes a rare instance of AAA causing rapid extrusion of a maxillary central incisor in a 17-year-old female. The patient presented with spontaneous pain and mobility of the extruded tooth, accompanied by localized swelling. Clinical and radiographic evaluations revealed pulp necrosis, an AAA, and apical bone rarefaction. Emergency treatment was initiated, including intracanal medication with calcium hydroxide and temporary splinting of the tooth. Subsequent treatment involved obturation with gutta-percha and sealer, followed by permanent restoration. Radiographic and clinical recalls up to 5 years demonstrated complete periapical healing, normal tooth mobility, and no recurrence of symptoms. Effective management, including timely root canal therapy and splinting, led to successful long-term outcomes. This case underscores the importance of prompt diagnosis and immediate, tailored treatment to manage AAA and prevent severe complications.

Keywords: Acute Apical Abscess; Healing; Pulp Necrosis; Rapid Extrusion; Splinting

Introduction

An acute apical abscess (AAA) is an inflammatory response to the pulp necrosis/infection, characterized by rapid onset, spontaneous pain, tooth tenderness to pressure, pus formation, and associated swelling of the surrounding tissues [1, 2]. The affected tooth may experience pain upon pressure or biting, and varying degrees of mobility may be observed [2]. Due to the acute nature of the condition, periradicular bone loss is not always visible in radiographs. AAA typically arises from pulp necrosis caused by caries or dental trauma, where micro-organisms invade the pulp and gain access to periradicular tissues through main apical foramen or lateral canals, leading to inflammation or apical pathosis [3, 4]. The purulent exudate resulting from the bacterial infection in the root canal may spread to the surrounding spaces, including maxillofacial regions and, in rare cases, cause tooth extrusion. In some instances, it can also lead to systemic complications and/or cellulitis [5], with periapical abscesses and toothaches being the most urgent non-traumatic conditions requiring dental intervention [6]. Host-related permanent/transient factors can influence the severity and progression of AAA [5], and specific immune responses, including specific antibodies against endodontic microorganisms can also play a role [7-9]. The virulence and pathogenicity of the infecting microorganisms significantly contribute to the development of symptoms and signs of periapical diseases [10]. Consequently, early intervention, such as root canal therapy or incision and drainage of the soft tissue, is critical in managing the condition and preventing further complications [11]. This case report discusses an unusual and a rare occurrence of rapid dental extrusion caused by an AAA.

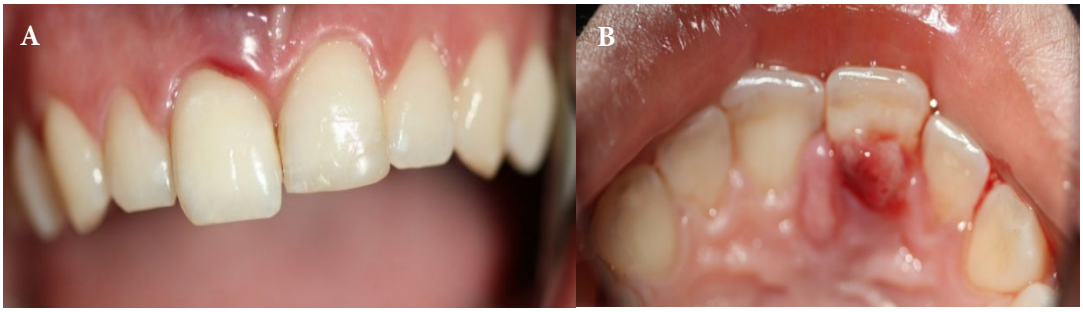

Figure 1. Labial and palatal views of the right central maxillary tooth with extrusion and granulation tissue swelling in the cervical region of the tooth, diagnosed with pulp necrosis and acute apical abscess

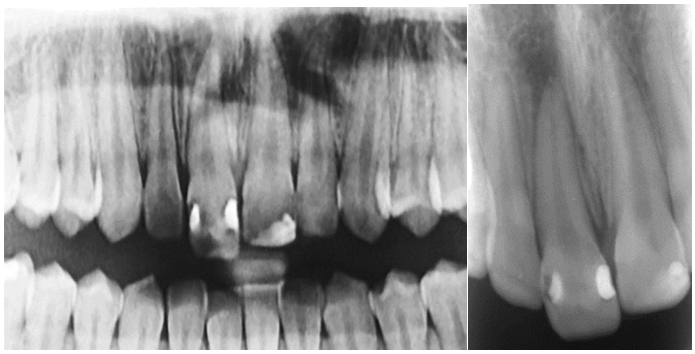

Figure 2. Initial panoramic and periapical radiographs of tooth #8

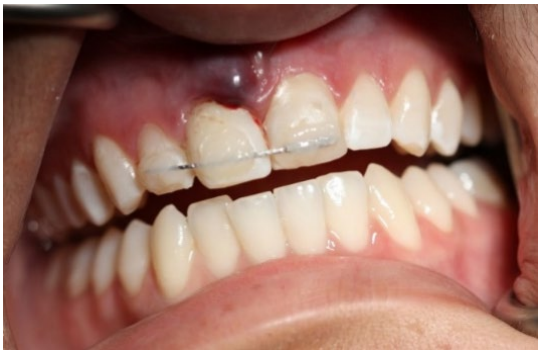

Figure 3. Splinting tooth #8 to the adjacent teeth with orthodontic wire and composite resin

Case Report

A 17-year-old girl attended the private office of an endodontist and reported that her anterior tooth had come out of its place since that morning, happening in only 4 h when she was at school. The patient had no systemic diseases and had no fever then, but she felt a general malaise. She reported no history of a traumatic accident on the anterior region of the jaws. She had spontaneous pain, with no previous swelling or pain on the extruded tooth. During the extraoral examination, some swelling was observed on the upper lip, with no asymmetry in the intraoral examination. Extrusion of the right central incisor was observed with local swelling and granulation tissue near the cervical region in both the buccal and palatal gingival soft tissues (Figure 1). The tooth exhibited grade III mobility. It had tooth-colored restorations on both the mesial and distal proximal aspects, with no tooth discoloration. The patient had noticed that her tooth was displaced rapidly out of its socket. She had asked her parents to take her to the dental clinic near the school. The dentist in the dental clinic ordered a panoramic radiographic examination and had referred her to the endodontist's office. The endodontist ordered a periapical radiographic examination to assess the surrounding area properly (Figure 2). There was bone rarefaction in the apical region of tooth #8, with 3 mm of tooth displacement on the periapical radiograph. Pulp sensibility tests were performed. The tooth did not respond to cold test (FT, Diamant, feiz Teb, Iran) Electrical pulp test (Parkell, Edgewood, NY, USA). The tooth was painful in the palpation of soft tissues around it.

The symptoms and signs of the patient and radiographic interpretations led to a diagnosis of tooth necrosis and AAA. The treatment plan was discussed with the patient and her parents, including initial root canal treatment and replacement of the tooth into the socket, followed by splinting it to the adjacent teeth and follow-up of the patient for adjunctive therapies if indicated. The prognosis of the treatment was explained to the patient who accepted the treatment plan, and an informed consent form was signed by the patient's parents.

The treatment began because of the urgent situation. After infiltration injection with 2% lidocaine containing 1:80000 concentration of epinephrine (Daru Pakhsh, Iran), an access cavity was prepared, and working length was determined with Root ZX apex locator (J. Morita, Kyoto, Japan). The working length was confirmed with a #15 K-file (Mani, Tochigi, Japan) and periapical radiographic imaging. Root canal preparation was carried out with copious irrigation using 5.25% NaOCl using the crown-down technique with K-files (Mani, Japan) and #3 and #2 Gates-Glidden drills (Mani, Tachigiken, Japan).

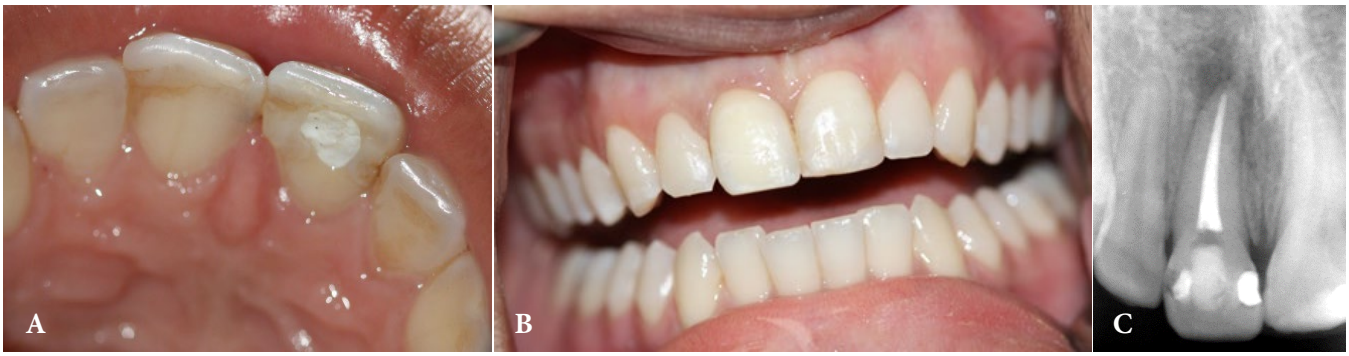

Figure 4 *.A)* The second session; completing root canal therapy of tooth #8 and splint removal; *B)* Tooth restoration ; *C)* Periapical radiography immediately after root canal obturation

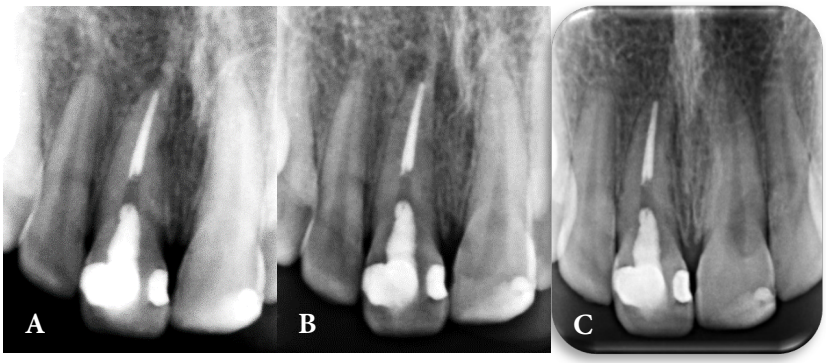

Figure 5. Periapical follow-up radiographs of tooth at *A)* 6-month; *B)* 14-month; *C)* Five-year

Calcium hydroxide (Golchadent, Tehran, Iran) was used as an intracanal medication because of the severity of the pain. Then the access cavity was sealed with Coltosol (Ariadent, Tehran, Iran) temporary restoration. The tooth was splinted to the adjacent teeth with an orthodontic wire and composite resin (Figure 3).

The occlusion was checked in the centric relationship, and premature contacts were removed by a high-speed fissure bur. The patient was advised to avoid eating hard food for a week with her anterior teeth. Ibuprofen (400 mg; Jaber Ebne Hayan, Iran) was prescribed as a *Pro Re Nata* (*PRN*) (when necessary) analgesic [12]. Since the patient was feeling malaise, and the upper lip was swollen Amoxicillin (500 mg; Farabi, Iran) was prescribed *tid* (three times a day) for 3-4 days as a drug of choice [13]. In the next treatment session nine days later, calcium hydroxide was washed out from the root canal with 5.25% NaOCl and 17% EDTA (Ariadent, Iran). Instrumentation was carried out with #30 K-file (Mani, Japan), and obturation was completed with gutta-percha (Meta Biomed, Cheongju, Korea) and sealer AH-26 (Dentsply, DeTrey, Konstanz, Germany) using the lateral condensation technique. The patient's pain during palpation and percussion reduced significantly, with no swelling at the upper lip. The splint was removed, and tooth mobility was Grade I. Therefore, the patient was referred to a dentist for tooth restoration (Figure 4). In the follow-up sessions after 6 and 14 months, periapical healing was detectable on radiographs and in clinical examinations (Figures 5A and B). After five years of follow-up, complete healing of the periapical lesion was seen in the periapical radiographic examinations, with no pain on palpation and percussion; the mobility was in the normal range, and the tooth was asymptomatic (Figure 5C).

Discussion

The present case report highlights an unusual manifestation of AAA, namely rapid tooth extrusion in a young girl. A similar case of extrusion occurring within three days has been reported [14]. The authors attributed this to the periodic acute inflammation damaging the periodontal ligament, leading to tooth extrusion. However, in the current case, extrusion occurred in just a few hours, likely due to the pressure from the apical abscess. This patient had chronic apical periodontitis, with radiolucency visible at the apical region, and the

exacerbation of this condition led to significant tooth extrusion due to periodontal ligament destruction.

Acute apical abscess might cause various complications. The infection can spread to the maxillofacial spaces, cause mortality, and potentially become a life-threatening problem [9, 15]. A case report describes retrograde bacterial spread to adjacent teeth from an apical lesion [16]. Pus aggregation at the apical region of the tooth after pulp necrosis and infection can occur. The involvement of facial spaces is related to the length of the tooth root and the position of the muscle attachments to the bone and cortical plate thickness. The pus can discharge through the cortical plate and cause soft tissue swelling in the buccal or palatal/lingual vestibule or create a pathway through the gingival sulcus. In an uncommon situation, the pus aggregation might be aggravated, extruding the affected tooth. The cause of this situation might be the pressure of the apical abscess [17].

Bacterial infection in the AAA is a polymicrobial consortium, and individual species might cause different symptoms and complications compared to when they are in association with other bacteria or microorganisms. Multiple microbial biofilms are necessary to form acute or chronic apical periodontitis [18]. Therefore, the ability of the bacterial community to invade the surrounding tissues and virulence factors are essential to forming an acute disease, such as AAA [19]. On the other hand, many host-related factors, such as systematic conditions (*e.g.*, diabetes, herpesvirus infection, stress, autoimmune diseases) [20-22] and gene polymorphism, can predispose individuals to develop acute infections [23].

Treatment of AAA is the incision for drainage from the root canal or soft tissue in cases with soft tissue swelling. One study showed that incision and drainage could produce improvements in a shorter time than when drainage is only from the root canal space, and in most patients with AAA, clinical improvement is achieved after 72 h [24]. According to the Position Statement of the European Society of Endodontology, when swelling is localized, and the patient's systemic condition is satisfactory, systemic or adjunctive antibiotic use is unnecessary, and analgesics can be prescribed for pain control [25].

Sometimes the acute responses of the tooth might appear after some time with chronic apical periodontitis, exacerbating after a while. Therefore, radiolucency and rarefaction can be observed on radiographic images.

The reason for pulp necrosis possibly was leakage around the composite restoration at both mesial and distal aspects of the crown. It was reported that restorations with the potential of leakage are more harmful to the pulp than unfilled prepared cavities [14] because of the chronic nature of pulp necrosis followed by leakage around the restoration.

Asymptomatic apical periodontitis was evident in the periapical radiography, and due to its asymptomatic nature, the patient was not aware of the problem. After a while, exacerbation occurred, causing tooth mobility and extrusion due to the apical pressure of the lesion [26]. In this case, because of the rapid onset of the extrusion of the tooth from its socket, repositioning of the tooth in the socket and functional splinting for a couple of days is considered an essential component of the treatment plan after pulpectomy and drainage of pus from the root canal system. The application of splints for traumatic dental accidents is widely accepted [27-29], but there is no evidence about tooth splinting after tooth extrusion followed by an AAA [14]. It has been reported that intentional replantation can be used to treat a tooth with extrusive luxation [27]. The concept of splinting an extruded tooth is based on immobilization and support of the tooth in the correct position to reestablish the periodontal ligament attachment [26]. It can be considered an adjunctive part of the treatment plan in a tooth with extreme tooth displacement in a particular situation rather than traumatic injuries. A 5-year follow-up, in this case, showed that the tooth after this treatment was functional and asymptomatic.

Conclusion

A 5-year follow-up of the patient with AAA and extruded teeth by splinting of the tooth for 10 days was presented in this case. There is no evidence of splinting an extruded tooth after an apical abscess and complete healing for a long time. However, further studies with long-term follow-ups are necessary to treat patients in the best manner possible.

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Authors' contributions
Conceptualization: MP, Methodology: AS, Formal analysis and investigation: MP/HM/AS, Writing-original draft preparation: HM Writing-review and editing: HM, Supervision: MP, All authors read and approved the final manuscript.

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