



CASE REPORT

Atypical facial pain related to apical fenestration and overfilling

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Abstract

Pasqualini D, Scotti N, Ambrogio P, Alovise M, Berutti E. Atypical facial pain related to apical fenestration and overfilling. *International Endodontic Journal*, **45**, 670–677, 2012.

Aim To report a case of apical fenestration and overfilling in which unusual pain characteristics made differential diagnosis challenging.

Summary A 32-year-old woman with diffuse, spontaneous, moderate pain in the maxillary left posterior sector, exacerbated by masticatory and facial muscle movement, with intense sporadic electric-shooting pain, underwent clinical examination and 3D cone beam computed tomography (CBCT). Apical fenestration with protrusion of the mesial root of tooth 26 beyond the buccal cortical plate, extrusion of canal filling material into the soft tissues and a periosteal reaction were detected. Surgery was performed under the operating microscope. The filling material and surrounding fibrous tissue were located, dissected from healthy soft tissues and removed. The mesiobuccal root apex was resected with a bur to within the bony crypt. A root end was prepared and filled with Tech Biosealer RootEnd™ (Isasan, Como, Italy). At the 2-week recall, the patient had complete resolution of the symptoms and good soft-tissue healing. The 1-year recall examination and intra-oral radiography confirmed complete resolution of the symptoms and health of periradicular tissues.

Key learning points

- Apical fenestration may occur in 9% of cases and may be considered an anatomic predisposing factor for persistent pain after root canal treatment.
- This complication provides a considerable differential diagnostic challenge and is often misdiagnosed and mistreated.
- When correctly diagnosed through an accurate, multidisciplinary approach, it may be managed with a simple surgical procedure in which the endodontist should play a key role.
- Misdiagnosis and over treatment of apical fenestration, through the surgical management of chronic facial pain conditions, could lead to severe exacerbation of chronic pain, which may potentially become persistent or, indeed, intractable.

Keywords: apical fenestration, apical surgery, atypical pain, operating microscope, surgical endodontics.

Received 6 September 2011; accepted 15 January 2012

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Introduction

Dental pain is the most prevalent form of oral-facial pain, affecting approximately 12% to 14% of the population over a 6-month period (Locker & Grushka 1987). The majority of patients experiencing oral-facial pain have symptoms caused by dental-related pathosis and odontogenic pain mechanisms, which may be successfully diagnosed and treated. However, in clinical practice, endodontists are often faced with difficult clinical cases, where differential diagnosis is complicated and it is difficult to determine whether pain in the oral-facial region is attributable to odontogenic or nonodontogenic causes. This can be challenging, because pain of endodontic origin and nonodontogenic pain may have similar characteristics, because of common features of the symptoms reported by the patient, and uncertain clinical-radiological information.

Alveolar fenestration is defined by the American Association of Endodontists (AAE) (2007) as a window-like opening or defect in the alveolar plate of bone, frequently exposing a portion of the root, usually located on the facial aspect of the alveolar process. No involvement of the marginal bone is present. Differences have been observed amongst ethnic groups and genders. However, the overall prevalence ranges from 0.23% to 16.9% (Larato 1970, Abdelmalek & Bissada 1973, Davies *et al.* 1974, Goldstein *et al.* 1976, Urbani *et al.* 1991). Studies on human skull collections report a mean prevalence of 9% (Jorgic-Srdjak *et al.* 1998, Rupprecht *et al.* 2001), the mesiobuccal root of the first maxillary molar being the most frequent (37%). These findings have been confirmed by a recent cone beam computed tomography (CBCT) analysis on patients with periradicular defects of endodontic origin (Yoshioka *et al.* 2011). Type V defects, particularly buccal bone fenestration and exposure of the apical foramen (type V-1) or the apical one-third of the root (type V-2), were found in 4% and 5% of cases, respectively.

Apical fenestration is usually asymptomatic and has no clinical relevance. However, persistent pain may occur after root filling (Boucher *et al.* 2000). Even slight overfilling may irritate the periosteum and the overlying mucosa; however, no data are available on the prevalence of nonsymptomatic overfilling in this anatomic condition. Normally, the tooth is spontaneously sensitive only occasionally, and pain is usually perceived during palpation of the area and masticatory movements. In this report, a case of apical fenestration and overfilling is described that made the differential diagnosis challenging because of the unusual pain characteristics.

Case report

A 32-year-old woman presented for consultation at the University of Turin Dental School, Department of Endodontics, Turin, Italy, complaining of diffuse, spontaneous, moderate pain from the maxillary left posterior sector, exacerbated by masticatory and facial muscle movement, with intense sporadic electric-shooting pain in the area of the zygomatic muscle, for the past 3 months.

The patient's medical history was normal, but she reported previous consultations with a neurologist and a gnathologist, as suggested by her dentist. The first tentative diagnosis was 'atypical facial pain' of unspecified aetiology, after exclusion of trigeminal neuralgia through clinical examination, with no instrumental tests or drug trial, and further investigations were requested for a more precise diagnosis. The patient underwent a specialist examination at the Orofacial Pain Unit of the University of Turin Dental School. Myofascial pain was excluded after clinical examination consisting of bilateral palpation of masseter, internal and external pterygoids, temporal and digastric muscles, with standardized pressure. The patient was then referred to the Department of Endodontics at the University of Turin Dental School. The patient confirmed that she had root canal

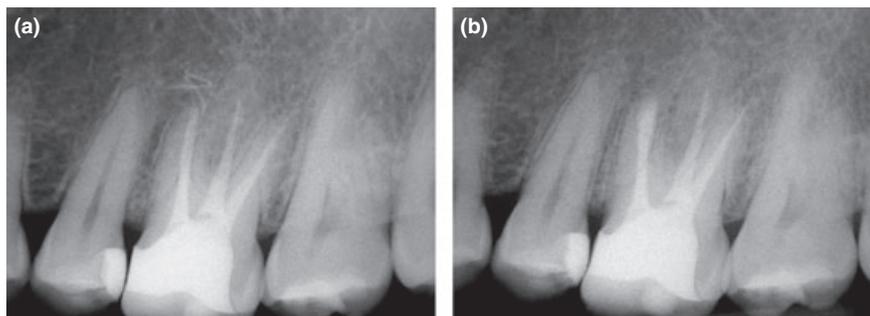


Figure 1 (a) Preoperative radiograph of tooth 26; (b) One-year follow-up.

treatment (RCT) on tooth 26 eighteen months previously. Pain-related symptoms were confined to the immediate post-treatment period. She also reported moderate caries and routine professional oral hygiene sessions. The patient's sensitivity was tested with stimuli delivered on the cheek, both on the affected and contralateral sides, consisting of slight touch of the skin and pressure exerted on the deep tissues.

Cutaneous stimuli did not produce any abnormal sensation, such as hyperalgesia or allodynia, on either side. Pressure exerted on various areas on both sides of the cheek did not produce pain, except for the area overlying tooth 26.

Intra-oral examination revealed normal periodontal probing depth; palpation in the apical area of tooth 26 provoked tenderness, whilst percussion was negative, and pulp sensibility tests on all the other teeth were normal. The restoration in tooth 26 was intact.

Traditional intra-oral radiography (Fig. 1a) revealed root canal overfilling and slight periodontal ligament (PDL) enlargement on the mesial buccal root, with no evident signs of lesions of endodontic origin (LEO). 3D CBCT (110 Kvp, 2 mA; NewTom VG, QR Verona, Italy) revealed apical fenestration with protrusion of the mesial buccal root of tooth 26 beyond the buccal bone plate, extrusion of canal filling material into the soft tissues, and a moderate periosteal reaction (Fig. 2). CBCT also confirmed the absence of any LEO on the palatal or distal roots. The patient was thus scheduled for apical microsurgery on the mesiobuccal root to remove excess material and the expected fibrous tissue (Fig. 3a) and to resect the protruding root apex back within the buccal aspect of the bone (Fig. 3b).

Surgery was performed under microscopic vision (OPMI Pro Ergo, Carl Zeiss, Oberkochen, Germany). After local anaesthesia with Mepivacaine 2% with adrenaline 1 : 100 000 and further haemostasis with Lidocaine 2% with adrenaline 1 : 50 000, a mucoperiosteal flap was elevated. The filling material and the surrounding fibrous tissue were located, dissected from the healthy soft tissues and removed. The mesial root apex

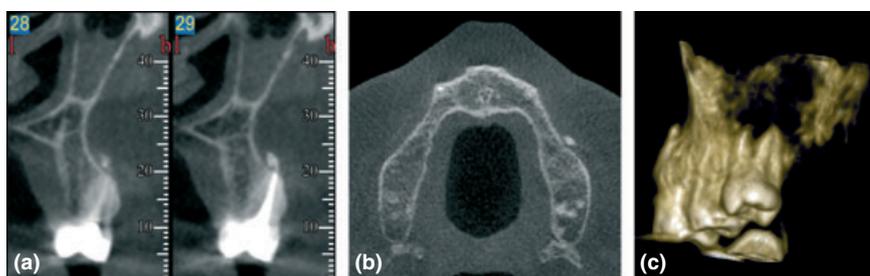


Figure 2 (a) Cone beam computed tomography cross-sectional and (b) horizontal axial views showing apical fenestration, overfilling and periosteal reaction; (c) 3D reconstruction of the volume, clearly confirming previous findings and their relation to surrounding structures.



Figure 3 (a) Intraoperative view of the extruded filling material surrounded by fibrous adhesences; (b) Intraoperative view of the resected and obturated root apex; (c) Two-week follow-up view showing good soft-tissue healing.

was then resected with a bur within the bony crypt. A root end was prepared with ProUltra™ surgical ultrasonic tip (Dentsply Maillefer, Ballaigues, Switzerland) and filled with Tech Biosealer RootEnd™ (Isasan, Como, Italy). The flap was then repositioned, sutured with nonresorbable 5-0 Tevdek™ (Deknatel; Teleflex Medical OEM, Tuttlingen, Germany) and gently compressed with gauze. Postoperative management consisted of ice packs, soft diet, optional analgesics and 0.12% chlorhexidine mouth-rinses twice daily for 2 weeks. After suture removal at 1 week, the patient still felt moderate discomfort. At 2-week recall, she reported complete resolution of the symptoms and had good soft-tissue healing (Fig. 3c). One-year recall examination and intra-oral radiography (Fig. 1b) confirmed complete resolution of symptoms and stable health of the periradicular tissue.

Discussion

Apical fenestration is an anatomic condition, which may occur in 9% of cases and may be associated with persistent pain after root filling (Boucher *et al.* 2000). This phenomenon was first described by Spasser & Wendt (1973) and then further analysed by Patterson (1981) and Weine & Bustamante (1995). The pain symptoms cannot be attributed to the bone defect itself, as teeth that have not undergone root canal treatment are generally asymptomatic. Pain has been attributed to mechanical irritation of the overlying periosteum and mucosa, caused by contact with the extruded filling material (Boucher *et al.* 2000). Affected teeth do not usually have any sensitivity. Spontaneous pain is rare, and pain is usually elicited by local intra-oral palpation and by movements of the masticatory or facial muscles. These symptoms may be worsened by patients frequently rubbing the affected area with their fingers.

The clinical case described here revealed some unusual pain patterns, which could lead to initial misdiagnosis. Moderate spontaneous pain was present and its exacerbation, caused by movements of the masticatory and facial muscles, was often associated with electric-shooting pain. Neurological examination could not identify the origin of pain that was diagnosed as 'atypical facial pain'. Furthermore, the patient failed to correlate the onset of painful symptoms to the previous endodontic treatment, as a relatively symptom-free interval of eighteen months had intervened. It may be hypothesized that the overfilling led to a subsequent chronic reaction, with fibrous encapsulation of the material over time, rather than to the transient post-treatment pain reaction. Chronic inflammation may have initially adapted to the irritant, without causing significant pain or swelling. In this case, however, new irritants, as well as mechanical stimulation of the fibrotic adhesences and of the overlying periosteum and mucosa, may have stretched nearby nerve terminals, which could explain the unusual electric-shooting neuralgia-like symptoms.

The first tentative diagnosis was 'atypical facial pain' (ATFP) of unspecified aetiology. ATFP is defined by the recent revision of the international classification of headache disorders by the International Headaches Society (IHS), as persistent idiopathic pain. The

pain is described as 'persistent facial pain that does not have the characteristics of cranial neuralgias and is not attributable to another disorder'. The diagnosis of ATRP is not an easy task and is usually a process of elimination (Agostoni *et al.* 2005). The aetiology remains unknown and different neuropathic mechanisms may be involved, including nociceptor sensitization, phenotypic changes and ectopic activity from the nociceptors, central sensitization, possibly maintained by ongoing activity from initially damaged peripheral tissues, sympathetic abnormal activity, alteration of segmentary inhibitory control and hyper- or hypo-activity of descending controls (Woda & Pionchon 2000).

It is well known that clinical pain may manifest itself in several forms. Spontaneous pain, in the absence of persisting noxious stimuli, may reflect central or peripheral changes in pain signalling. Numerous studies carried out on animal models, and humans have clearly demonstrated that pain hypersensitivity originates from a condition of long-lasting nociceptive activity able to produce neuroplastic changes both in nociceptors and in spinal cord neurons (Koltzenburg 1995, Coderre & Katz 1997, Julius & Basbaum 2001). This implies long-lasting or permanent alterations in nociceptive signal transmission and processing, that is, a condition that is referred to as 'chronic pain'. Tissue inflammation and several forms of nerve injury may be the crucial events that generate chronic pain, defined as inflammatory and neuropathic pain, respectively. Inflammatory nociceptive pain is associated with tissue damage and subsequent inflammatory processes. It is adaptive and it elicits physiologic responses through healing processes. Neuropathic pain is caused by damage to neurons in the peripheral or central nervous systems, with sensitization to one system. Other factors, such as emotional- and stress-related factors may modulate nociception, through sympathetic nervous system activity. Postganglionic sympathetic fibres enhance neuropathic and inflammatory pain in several areas of the body in humans and animal species. This occurs through an action exerted on nociceptors and/or on its afferent fibres (Janig & Habler 2000, Passatore & Roatta 2006).

Clinical symptoms may differ depending on the mechanism by which pain becomes chronic and they may assist in differential diagnosis. Symptoms such as tenderness, hyperalgesia and allodynia (pain sensation evoked by usually innocuous stimuli, such as touch) may be observed on both injured and contralateral sides. They tend to signify the occurrence of a peripheral, and possibly also central (for allodynia), plasticity (Woolf & Costigan 1999, Woolf & Salter 2000).

The patient reported here did not exhibit signs of hyperalgesia in areas distant from the affected point, neither did she exhibit allodynia. On this basis, it was hypothesized that relevant plastic changes in nociceptive signal transmission and processing had not taken place. If this were the case, surgical intervention at the peripheral level would not have abolished the pain. Rather, the electric-shooting, neuralgia-like pain occurring when the patient contracted her masticatory muscles, and the presence of periosteal reaction with fibrotic adhesions visualized at the CBCT 3D examination, suggested that nearby nerve terminals were stretched by movement or palpation. The positive outcome of the surgery appears to confirm this hypothesis.

Pain related to masticatory muscle movements led the patient's dentist to hypothesize nonodontogenic dental pain of musculoskeletal origin and to suggest the diagnosis of 'trigger point in myofascial pain'. Indeed, several muscles, including masseter, temporal and digastric muscles, may refer pain to teeth (Travell & Simmons 1983). Myofascial pain is usually described as steady, aching and deep, and diffuse or referred, but it may be exacerbated by the palpation of trigger points or muscles, and only occasionally localized. Differential diagnosis with odontogenic pain is based on the observation of diffuse pain, exacerbated by trigger points (in myofascial pain) or muscle palpation, pain during masticatory movements and possible limitation of jaw movement. The pain is not relieved by local anaesthesia and is not influenced by intraoral thermal stimuli. Pain may

occasionally be referred. Pain in the upper portion of the superficial layer of the masseter may refer towards maxillary posterior teeth, whilst pain in the lower portion refers towards mandibular posterior teeth. Pain in the anterior belly of the digastric refers towards mandibular anterior teeth and that of the temporalis refers towards maxillary anterior and posterior teeth (Travell & Simmons 1983). Molars are the teeth most frequently subjected to referred pain from muscle or trigger point palpation. The masseter muscle is the major cause of referred pain to teeth. Palpation of masseter, temporalis, digastric and lateral pterygoid muscles, as well as the temporo-mandibular joint, is the main step towards differential diagnosis and the exclusion process between odontogenic and nonodontogenic pain of suspected musculo-skeletal origin (Travell & Simmons 1983).

Initial radiographic investigation by traditional two-dimensional intra-oral radiography was unable to detect a defect localized along the buccal-lingual projection; 3D low-dose CBCT analysis was thus crucial to formulate a diagnosis of apical fenestration. The importance of 3D imaging for appropriate diagnosis of oral-maxillofacial diseases is increasingly recognized, and the superior diagnostic accuracy of this technique compared to two-dimensional imaging has been clearly documented (Stavropoulos & Wenzel 2007, Estrela *et al.* 2008). In endodontics, the advantages associated with 3D imaging are becoming increasingly evident, and this approach is particularly indicated to visualize tooth anatomy and its relation to the surrounding tissues (Nair & Nair 2007), as is shown in this case report.

However, some crucial ethical issues were involved in the postoperative documentation of the case. A second CBCT investigation after complete resolution of the symptoms and intra-oral radiographic documentation of healing would have caused considerable concern. As recommended by the AAE and AAOMR (2010) Joint Position Statement on the *Use of Cone-Beam-Computed Tomography in Endodontics*, CBCT should be limited to particular conditions, over-exposure to radiation should be avoided, and in no case may the technique be considered 'routine' or utilized for screening purposes. Therefore, clinical and radiological follow-up with traditional intra-oral examination were considered appropriate. Again for ethical considerations, further studies employing 3D CBCT to investigate the prevalence of nonsymptomatic overfilling in such anatomic conditions may be considered questionable. Actual data can thus only be extrapolated from case reports, descriptive studies on human skulls (Jorgic-Srdjak *et al.* 1998, Rupprecht *et al.* 2001) or retrospective CBCT investigations (Yoshioka *et al.* 2011) for other clinical-pathological purposes.

To treat persistent pain because of apical fenestration, surgical resection of the protruded root apex within the buccal bone level is required. The bone may undergo spontaneous repair and the pain be completely resolved (Boucher *et al.* 2000). The mucoperiosteal flap also enables the area to be explored and can confirm the diagnosis. Mineral Trioxide Aggregate (MTA)-based cements or Super-EBA cements are currently indicated as root-end filling materials (Kim & Kratchman 2006), although more favourable outcomes have been obtained with MTA (Baek *et al.* 2010). This latter procedure was used in the surgical management of the case presented here. The excess filling material was found to be extensively extruded into the surrounding soft tissues, with periosteal reaction and fibrous encapsulation of the filling material. The subsequent fibrous adhesences probably caused further irritation and triggered the electric-shooting-like pain symptoms. Surgical removal of the irritating filling material and of the fibrous adhesences leads to soft-tissue healing and complete resolution of the pain within 2 weeks.

Conclusions

Apical fenestration, although often misdiagnosed, may be considered an anatomic predisposing factor for persistent pain after root canal treatment. It may cause unusual

neuralgia-like pain features, as in the case presented here which is rare and should be treated with caution. Because of the numerous mechanisms potentially involved in chronic pain of the maxillofacial area, a multidisciplinary approach is necessary. Differential diagnosis between odontogenic and nonodontogenic pain is required, as persistent and chronic pain are more common in the head and neck region than in any other part of the body (Shankland 2001). Over-diagnosing apical fenestration and, in so doing, initiating the surgical management of chronic facial pain conditions could severely exacerbate chronic pain, which may become persistent or, indeed, intractable.

Conflicts of interest

The authors declare no competing conflicts of interest with the materials discussed in this manuscript.

Acknowledgements

We gratefully thank Professor Magda Passatore, Department of Neurosciences, Physiology Division, University of Turin Faculty of Medicine, for her cooperation and friendship.

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Supporting information

Additional Supporting Information may be found in the online version of the article:

Movie S1. Video clinical case presentation. The video contains a brief background presentation to the topic reported in this case study. The clip also includes pre- and post-operative information, together with microscopic views of surgical intervention. To aid comprehension, the authors suggest that the video is viewed once the article has been read.

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