Diagnostic Challenges of Neuropathic Tooth Pain

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Abstract

This article presents the clinical characteristics, epidemiology, pathophysiology and treatment of 2 neuropathic conditions: trigeminal neuralgia and atypical odontalgia. A case report highlights the complexities involved in diagnosing neuropathic pain. Neuropathic pain is chronic, diverse in quality, difficult to localize and it occurs in the absence of obvious pathology. To avoid multiple, ineffective dental treatments, general practitioners must be familiar with the signs of nonodontogenic sources of tooth pain.

MeSH Key Words: *diagnosis, differential; facial pain/diagnosis; facial pain/physiopathology; toothache/diagnosis*

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One of the most challenging and rewarding aspects of general practice is the diagnosis and treatment of pain. An estimated 22% of the general population experiences orofacial pain in any given 6-month period.[1] Furthermore, persistent and chronic pain is more prevalent in the head and neck region than in any other part of the body.[2]

Misdiagnosis of orofacial pain is common. The convergence of sensory neurons to higher centres makes localization and interpretation of pain symptoms difficult.[3] Myofacial, neurovascular, sinus and cardiac structures can all be the source of referred dental pain, frustrating the diagnostic efforts of the general practitioner.[4]

Although, for the most part, tooth pain is resolved with endodontic treatment, in rare instances clinical response is not predictable and pain persists despite intervention. These cases may undermine both the patient's and dentist's confidence in clinical diagnosis and treatment. More important, the patient may undergo many other irreversible dental treatments, with no resolution of the pain symptoms.

The purpose of this article is to review the etiology, diagnosis and treatment of 2 conditions that may mimic dental pain: trigeminal neuralgia and atypical odontalgia. A case report is presented to illustrate the complexities of diagnosing and treating orofacial pain.

Case Report

A 64-year-old woman was referred for assessment of enigmatic pain in her lower left teeth. She complained of shooting pain that started after a routine hygiene visit 4 weeks earlier. To resolve her discomfort, she recently had amalgam fillings replaced with bonded composite restorations in teeth 34 and 36 (**Fig. 1**). The patient was subsequently referred to me when the pain did not abate.

Clinical examination revealed that teeth 34 and 36 were heavily restored. The fillings were well sealed and the occlusal and interproximal contacts were adequate. No signs of gingival inflammation or pathology were present. The patient reported that tooth 35 was treated by root canal therapy 20 years earlier and the tooth had been crowned only 3 years ago. Percussion, palpation and bite testing of quadrant 2 and 3 were negative. As well, all teeth responded within normal limits for vitality when tested with cold. No noticeable pathologies were observed on the radiographs.

The patient had not experienced any spontaneous pain to that point. She directed us to observe that mechanical stimulation of the buccal root surface of tooth 36 resulted in a sharp "electric" shooting pain in the area. At that appointment, the root surfaces of tooth 36 were desensitized with a self-etch bonding system (Clearfil SE, Kuraray Co., Osaka, Japan). The patient was also given Sensodyne dentifrice (GlaxoSmithKline Inc., Pittsburgh, Penn.) with instructions for use.

The patient returned 2 weeks later reporting that she still had intermittent pain on the lower left and that tooth 36 was now sensitive to biting and chewing. Percussion and cold testing elicited a strong reaction in this heavily restored tooth. Root canal treatment was completed, on the assumption that irreversible pulpitis in tooth 36 was the source of her pain (**Fig. 2**).

On follow-up 1 week later, the patient was still experiencing what she described as "jolts of pain" from her lower left teeth. At this time she pointed to tooth 35, and insisted that sharp pain resulted when chewing or touching the buccal gingiva of tooth 35 with her toothbrush. Tooth 36

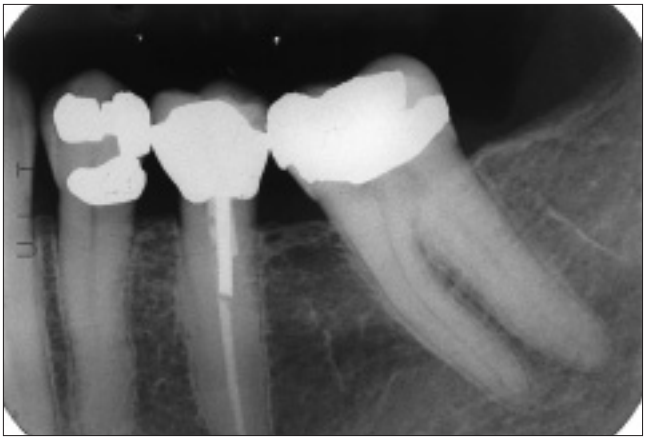

Figure 1: *Preoperative radiograph of quadrant 3 before restorative or endodontic treatment.*

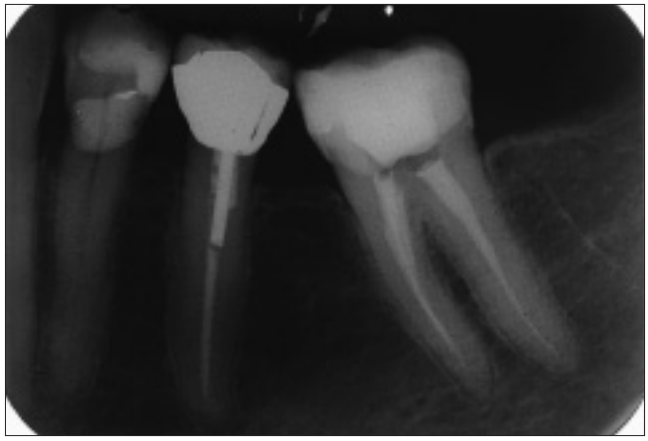

Figure 2: *Postoperative radiograph of tooth 36 root canal treatment.*

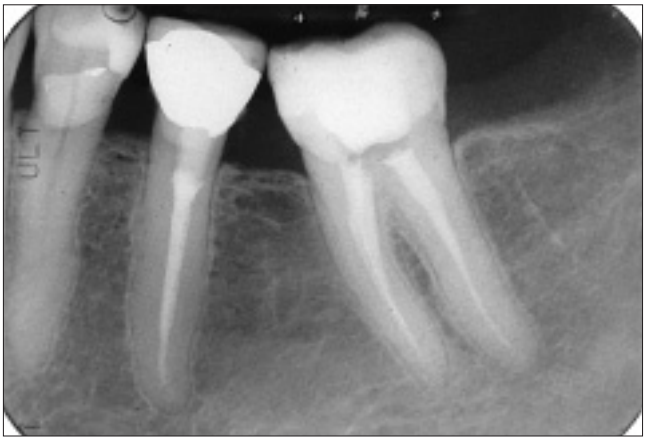

Figure 3: *Postoperative radiograph of tooth 35 root canal re-treatment.*

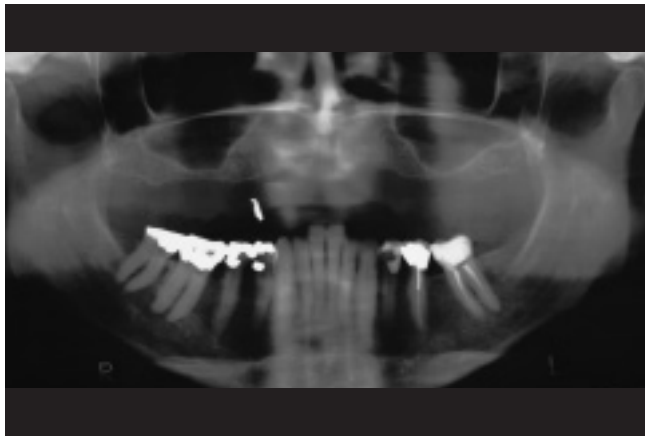

Figure 4: *Panoramic radiograph with no observable pathology.*

was still slightly sensitive to percussion; however, the result was not the stabbing pain the patient had been experiencing. The root surface of tooth 36 was no longer sensitive. She now insisted that the pain was from tooth 35 and, at this time, revealed that the tooth had "never felt right" since the root canal treatment 20 years earlier. The patient was about to leave on a month's vacation and pleaded with me to do something to relieve her discomfort.

To satisfy the patient, I reluctantly re-treated tooth 35 by root canal. It was noted at the time of the treatment that block injection resulted in cessation of the gingival pain. Standard root canal procedures were followed and, after filling the canal with gutta-percha, the porcelain-fused-to-metal crown was recemented (**Fig. 3**).

The patient returned to our office after her vacation and informed us that the stabbing pain had not resolved. We observed that palpating the buccal gingiva at tooth 35 would elicit pain. Palpating certain areas of the lower left vestibule and lip had the same result. Unsure that the pain was the result of a tooth problem, we referred the patient to an oral surgeon for consultation. He was able to see her within 2 weeks and concluded that, in the absence of any notable pathology (**Fig. 4**), the pain must be neuropathic in nature. We discussed 2 possible diagnoses: trigeminal neuralgia and atypical odontalgia.

The oral surgeon prescribed carbamazepine, 200 mg 3 times daily. The patient's symptoms resolved within 2 weeks. Against the surgeon's advice, the patient discontinued the medication after 5 weeks believing that the problem was solved. Fortunately, the pain did not recur.

This case illustrates the difficulties often encountered in diagnosing and treating orofacial pain. First, the patient's interpretation of symptoms and reaction to clinical testing can reflect both emotional and physical components of pain.[5] To further complicate the issue, a patient's interpretation of the discomfort may not truly reflect the area in which the pathology is present.[2,3] In this case, initial diagnosis was compromised by conflicting reports of the nature and source of the patient's pain, a common occurrence in neuropathic conditions. Second, response to treatment (i.e., medication) may be the only way to confirm diagnosis of neuropathic pain.[6] In this case, a positive response to anticonvulsant therapy supported a neuropathic basis for the symptoms. Last, despite careful examination of the symptoms and response to

treatment, differential diagnosis of neuropathic pain conditions can be challenging. A conclusive diagnosis of trigeminal neuralgia or atypical odontalgia was never reached. The diagnostic challenges encountered with this patient prompted further investigation into these 2 neuropathic conditions that often have dental components.

Trigeminal Neuralgia

The most common cause of facial neuralgia is trigeminal neuralgia, affecting 4–5 people per 100,000 population,[6,7] and more often affecting women over 40 years of age.[6–8] A genetic predisposition to the condition has not been found.[8] However, trigeminal neuralgia does occur in about 1% of patients with multiple sclerosis and 2% to 8% of patients with trigeminal neuralgia have multiple sclerosis.[7]

Trigeminal neuralgia is characterized by sudden, sharp, severe unilateral pain. It is often described as a stabbing, shooting, burning or paresthesia sensation.[9–11] The pain follows one or more branches of the trigeminal nerve.[6,9–12] It can last seconds to minutes, then disappear leaving pain-free intervals between attacks.[12] The paroxysms of pain may occur in rapid succession while the patient is awake, but they rarely occur during sleep.[6,9,10]

Trigger areas around the nose and mouth are a characteristic feature of trigeminal neuralgia. Attacks can be provoked by such innocuous stimuli as talking, chewing, tooth brushing or light touch.[9–11] The pain is often much greater than the stimulus. Local anesthetic placed in the trigger area reduces the pain, whereas a block may not.[4]

There are several theories regarding the mechanism of pain production in trigeminal neuralgia. All remain uncertain and controversial.[7] One theory suggests partial and focal nerve demylenation as a result of tumour or vascular compression. This can lead to abnormal transmission and processing of impulses along the trigeminal nerve.[6,7,13] Extensive use of magnetic resonance imaging (MRI) to document the presence of benign or malignant lesions, plaques of multiple sclerosis and proximity of vessels to the trigeminal nerve has supported this postulate.[10] Similarly, intraoral compression of the mental nerve by an ill-fitting denture can lead to trigeminal neuralgia-like symptoms.[10]

An alternative theory suggests that chronic irritation or trauma to the trigeminal nerve can cause ectopic action potentials and failure of segmental inhibition, leading to symptoms of trigeminal neuralgia.[14] In reality, for most patients with trigeminal neuralgia, there is no identifiable cause.[10]

Treatment goals have focused on prevention of pain. Commonly used drugs include anti-seizure/anti-epileptic medications such as carbamazepine, baclofen and phenytoin.[7,15,16] These drugs reduce neuronal excitability and discharge[16] and generally lead to relief from symptoms in 75% to 80% of patients within 24–72 hours.[6,8,15,16] Indeed, response to anticonvulsant treatment has been used as a diagnostic tool for trigeminal neuralgia.[6]

More recently, the use of topical capsaicin to block nociceptive fibres in the trigger area has shown some promise.[17–19] Unfortunately, data from large-scale testing of this therapy are not yet available.[17]

Finally, for cases in which nerve compression is the source of trigeminal neuralgia symptoms, microvascular decompression surgery can be effective. The procedure involves surgically removing vessel or tumour compression of the trigeminal nerve directly or indirectly via gamma knife radiosurgery.[6,7,13] In the future, MRIs will provide more accurate and well-validated diagnoses, in turn improving surgical treatment of certain forms of trigeminal neuralgia.[10]

Atypical Odontalgia

Atypical odontalgia, also known as idiopathic or phantom tooth pain,[20] was first reported by McElin and Horton in 1947.[21] This clinical condition has been validated extensively,[22–26] yet it is rarely reported.[27] It is usually characterized by persistent toothache following pulp extirpations, apicoectomy, or tooth extraction.[27] Facial trauma and inferior alveolar nerve block have also been found to cause atypical odontalgia.[23] Epidemiologic information indicates that 3% to 6% of patients develop atypical odontalgia after endodontic treatment.[28,29]

Characteristically, atypical odontalgia presents as prolonged periods of constant throbbing or burning pain in teeth or the alveolar process.[20,22–27] This is in the absence of any identifiable odontogenic etiology observed clinically or radiographically.[5] The pain is chronic; however, the patient's sleep is undisturbed, and there may be a brief symptom-free period on waking.[27] Patients often have difficulty localizing the pain.[22,23,27] It is usually worst at the site of the original trauma, but can spread to adjacent areas, unilaterally or bilaterally.[5,27] All ages can be affected, except for children; there is a preponderance among women in their mid-40s.[20,24–33] Molars and premolars in the maxilla are most often affected.[33,34] Local anesthetic block gives ambiguous results, and patients rarely find relief with analgesics, including narcotics.[23,27,35,36] Unfortunately, atypical odontalgia is often mistaken for a normal post-treatment or post-trauma complication.[27]

Although it is tempting to consider, psychological comorbidity has not been demonstrated in atypical odontalgia.[27] As in several chronic pain conditions, a high level of demoralization is evident. However, it is uncertain whether this is the cause or the effect of the condition.[27,35–38]

Many classification and diagnostic criteria for atypical odontalgia have been proposed.[39] However, it remains a diagnosis of exclusion after ruling out all other pathologies of the head and neck.[9,35,37] Patients often seek multiple endodontic or surgical treatments, realizing no relief or even exacerbation of their symptoms.[5,40] Accurate diagnosis depends on recognizing neurologic signs involving other teeth and nearby structures served by the same nerve.[9]

The pathophysiology of atypical odontalgia remains unclear. In 1978, Marbach[20] hypothesized that atypical odontalgia was of similar etiology to phantom limb pain.

Table 1 Differential diagnosis of odontogenic and neuropathic pain[a]

Odontogenic pain	Neuropathic pain
Pain is dull ache or occasionally sharp.	Pain may be dull, sharp, shooting or burning.
Response to stimuli, such as hot, cold or percussion, is predictable and proportionate.	Response to hot, cold or percussion does not reliably relate to the pain and may be disproportionate.
Pain is usually inconsistent and tends to get better or worse over time.	Pain is persistent and remains unchanged for weeks or months.
Pain often disrupts sleep.	Pain rarely disrupts sleep.
There is often an identifiable source (i.e., caries, deep restoration, periodontal disease, fracture line).	There is no obvious source of local pathology.
Local anesthesia of the suspect tooth eliminates the pain.	Response to local anesthetic is ambiguous.
	Pain may be felt in multiple areas or teeth.
	Repeated dental therapies fail to resolve the pain.

[a]*Adapted from Okeson.*[9]

Table 2 Differential diagnosis of trigeminal neuralgia and atypical odontalgia[a]

Trigeminal neuralgia	Atypical odontalgia
Pain is characterized as unilateral, paroxysmal and stabbing.	Pain is dull and continuous.
Trigger areas characterize pain.	Trigger areas occur less often.
More common after 40 years of age, peaking in the 50s and 60s.	More frequent in women in their mid-40s.
May occur in the absence of obvious trauma.	Usually precipitated by a traumatic event (root canal, extraction, etc.).

[a]*Adapted from Marbach and Raphael*[35] *and Okeson.*[9]

Deafferentation research has demonstrated that, after injury, organization and activity of central and peripheral nerves can change.[41–44] This can result in chronic pain and other related symptoms (paresthesia, dysesthesia).[41,43] For example, neuroma secondary to nerve trauma is thought to result in such pain.[9,20,24,25,27,32,34] Other mechanisms involved in the pathogenesis of pain include sensitization of pain fibres, sprouting of adjacent afferent fibres, sympathetic activation of afferents, cross-activation of afferents, loss of inhibitory mechanisms and phenotypic switching of afferent neurons.[41,43] These processes may underlie the clinical manifestation of atypical odontalgia.

Treatment of atypical odontalgia is similar to that of other neuropathic conditions. Tricyclic antidepressants (TCAs), alone or in association with phenothiazines, have been prescribed with good results.[5,20,22,30,31,35,36] Although these are mood-altering medications, their effectiveness is attributed to their ability to produce a low-grade analgesia in low doses.[9,36] Undesirable side effects require that TCAs be titrated to the lowest clinically effective dose and discontinued if pain symptoms abate.[45]

Topical application of capsaicin to painful tissue has also been investigated as a treatment for atypical odontalgia.[18,46,47] Pain reduction is achieved because C fibres depleted of substance P have a reduced ability to stimulate second-order neurons that relay pain signals to the central nervous system.[9]

Differential Diagnosis

Differential diagnosis of neuropathic pain conditions is the most challenging aspect of managing referred pain cases. Pain in the head and neck can be diverse. However, there are characteristics of odontogenic and neuropathic conditions that aid diagnosis (**Table 1**). Furthermore, although there is some overlap in clinical presentation, careful examination of symptoms can differentiate trigeminal neuralgia from atypical odontalgia (**Table 2**).

The fact that neuropathic tooth pain can present exclusively intraorally in the absence of obvious infection or trauma can be confusing to both patients and clinicians.[10] Patients in dental environments are more likely to be considered to have dental pain as opposed to patients referred to a neurologist. This is where patients' perception of their problem can influence treatment and referral considerations.[10] Careful history and clinical and radiologic examination are important. As well, thorough evaluations of the nature of pain, including aggravating and relieving factors and associated symptoms, aid correct diagnosis. Referral to a pain specialist or neurologist should be considered when conflicting reports occur and dental etiology is unlikely.

Conclusion

Neuropathic pain in the head and neck region is common and can result in multiple unnecessary dental treatments. Trigeminal neuralgia and atypical odontalgia are 2 neuropathic conditions that may compromise accurate diagnosis of orofacial pain. It is imperative that general practitioners recognize clinical characteristics of neuropathic pain to deliver appropriate therapy and avoid aggravating the condition. ◆

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