# Management Issues of Neuropathic Trigeminal Pain From a Dental Perspective

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*Neuropathic trigeminal pain conditions are more common than is generally appreciated. Sites inside the mouth as well as involvement of extraoral tissues are common manifestations of these disorders. There is a general lack of recognition of the complex characteristics of neuropathic trigeminal pain that frequently lead to mischaracterization of the nature of the complaint. Dentists are in an excellent position to detect the presence of neuropathic trigeminal pain and help to provide a rational diagnosis. The high prevalence of orofacial pain of dental origin and the dramatic similarities between neuropathic orofacial pain and odontogenic and other pathologic pains in the region frequently lead to incorrect diagnoses and, more importantly, inappropriate treatments that are frequently invasive and irreversible. The records of patients presenting with neuropathic pain at our university pain clinic were reviewed to gain insight into dental factors as they related to the etiology, presentation, diagnosis, and management of neuropathic pain of the trigeminal system. Relative to etiology, the records review revealed that most onsets were associated with a specific dental treatment or odontogenic symptom that resulted in a dental diagnosis or treatment. Initial treatment modalities that either caused the pain or were used to address painful symptoms commonly included replacement of restorations, endodontic therapy, apicectomy, extraction, splint therapy, and occlusal equilibration. Correct diagnosis, and particularly early definitive diagnosis, of neuropathic trigeminal pain is crucial to avoid invasive and potentially more damaging forms of treatment.* J OROFAC PAIN 2004;18: 374–380

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Neuropathic orofacial pain (neuropathic trigeminal pain) is a significant form of pain frequently encountered in dental and pain clinic settings.[1–4] The dentist is the health professional most likely to have first contact with patients who exhibit symptoms suggestive of neuropathic trigeminal pain, and dental treatments themselves have been implicated as a causal mechanism.

Chronic neuropathic trigeminal pain is a serious condition that almost always causes significant morbidity and suffering in those affected.[1,3,4] The presentation of a patient complaining of chronic moderate or severe orofacial pain will often result in the initiation of invasive and irreversible treatments directed at symptoms that are not responsive to traditional dental approaches; these treatments frequently have appreciable risk for increasing the severity of the pain and suffering.[2,3] While our understanding of basic neurophysiologic mechanisms associated with pain transmission and modulation has advanced substantially, our understanding of presenting clinical issues and appropriate treatment approaches is far less advanced.[5–9]

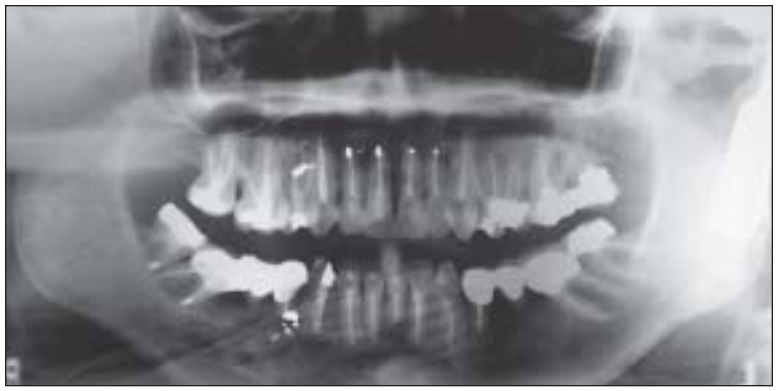

**Fig 1** Patient with chronic orofacial pain treated with multiple invasive attempts at eradication of symptoms.

Figure 1 illustrates an example of the clinical impact of misdiagnosed neuropathic trigeminal pain. This patient initially presented with pain diagnosed as odontogenic pain related to pulpal infection. Nonresolution of the original pain complaint led to additional invasive treatments, including further extensive root canal therapy, apical surgery of "nonresponding" teeth, extractions, eventual temporomandibular joint (TMJ) surgery followed by joint replacement and later by joint prosthesis removal. None of the treatments were successful, and it is unclear whether the chronic neuropathic trigeminal pain was the result of failed treatments or whether the initial symptoms were neuropathic. The unfortunate end result was the compounding of the pain disorder to all areas of the mouth. Pain of neuropathic origin or the role of behavioral and psychologic factors in the initiation and persistence of symptoms was never considered in the etiology or progression of symptoms and disability in this patient.

## Factors that Complicate Dental Assessment and Management of Neuropathic Trigeminal Pain

Regardless of the region of the body affected, the diagnosis and management of neuropathic pain are often difficult.[3,10,11] In the region of the oral cavity and face, assessment and management are even more difficult because a number of contributing factors singly or in combination confuse recognition of the disorder and inhibit definitive diagnosis.[12–16] The lack of randomized controlled trials of therapeutic interventions further complicates management.[2,3,5] The following is a partial list of complicating factors from a dental management perspective:

- The epidemiology of neuropathic trigeminal pain is unresolved.
- The nature of the initial complaint can be difficult to distinguish.
- Little is known about etiologic factors, including the nature of physical, chemical, microbial, and predisposing states.
- Various symptoms are manifested.
- A variety of clinical findings are associated with neuropathic trigeminal pain.
- There is frequently comorbidity with other conditions and symptom complexes, eg, temporomandibular disorders (TMD), pulp sensitivity, otolaryngologic complaints.
- Diagnostic approaches are not standardized.
- The management and treatment of neuropathic trigeminal pain, and the responses to that treatment, are highly varied.

Very little is understood about the epidemiology of neuropathic trigeminal pain; therefore, it is difficult to judge the scope of the problem and the burden it creates within society and individuals affected. The standard epidemiologic parameters commonly identified for other disorders have not been assessed in neuropathic trigeminal pain. For instance, the incidence and prevalence have not been determined, partially because clear case definitions for each type of neuropathic trigeminal pain have not been established.[3,4,13,17]

The apparent wide variation in clinical symptoms further clouds issues of diagnosis and management for all but the most obvious forms of neuropathic trigeminal pain, such as classical trigeminal neuralgia. Persistent pain after nerve resection is not difficult to attribute to neuropathic defects, but persistent pain after endodontic or periodontal therapy is less obviously neuropathic, and normally a diagnosis of neuropathic trigeminal pain is not considered a reasonable alternative diagnosis until all other physical and pathological diagnoses are excluded (eg, root fracture, endodontic failure, residual infection, occlusal disharmony).

Some of the exact symptom reports that would make a diagnosis of neuropathic pain probable in other regions of the body are often confused with local dental pathology (compare pain provocation upon light tactile stimulation of the skin to pain provocation from light tactile stimulation of a tooth).[1,10,13] The fact that certain clinical findings are common to both neuropathic trigeminal pain and other dental disorders also adds confusion to management.[1,11,18] For example, percussion of the teeth and temperature hypersensitivity are considered cardinal signs of local dental pathology or failed dental treatment, but similar responses to heat and percussion are often evident in patients with neuropathic trigeminal pain. Another confounding problem is the high degree of comorbidity with other painful and symptomatic orofacial disorders. The patient with symptoms suggestive of neuropathic trigeminal pain has a reasonable probability of also experiencing symptoms of other episodic or persistent painful orofacial and dental conditions.[2,18] For example, the prevalence of painful TMD approaches 20% in some adult populations. This represents a risk of 1 in 5 patients presenting with neuropathic trigeminal pain also exhibiting signs and symptoms of TMD; the TMD symptoms could lead clinicians to focus on the more prevalent disorder and believe it to be responsible for all components of the complaint, resulting in therapy that fails to address all aspects of the patient's distress.

Additional complications occur because neuropathic trigeminal pain may lead to changes in jaw posture and behaviors that initiate the onset of muscle pain.[18,19] Lack of a standardized approach to more common facial and oral pains is well recognized, as is the lack of definitive diagnostic methods and confirming tests. While some neuropathic trigeminal pain states have been rather well characterized and diagnostic methods defined,[1,3] other forms of neuropathic trigeminal pain not associated with surgery or trauma are poorly characterized from diagnostic and testing perspectives useful in dental and medical practice. In other aspects of health care, treatment outcomes are often used to assess the vigor of the diagnosis, but the lack of standardized treatments for neuropathic trigeminal pain and the high degree of variability in responses to treatment make confirmation of diagnosis through observation of treatment response much more problematic. Consistent responses to therapeutic interventions have not been seen except for responses to medication and surgical protocols for classic trigeminal neuralgia.[3–5,20]

Other confounding areas in the management of neuropathic trigeminal pain include the wide range of triggering factors along with minimal understanding of "at risk" factors for development of neuropathic trigeminal pain. In some patients, major structural and surgical injury to the tissues results in minimal if any residual neuropathic pain; in others, an extremely minor procedure (eg, anesthetic infiltration) initiates the onset of neuropathic trigeminal pain that persists for years and defies treatment interventions. This wide range of initiating factors and lack of knowledge of predisposing factors leaves the clinician with an inability to determine the value of risk-reducing strategies. It is also critical to recognize that behavioral status and behavioral response to severe or persistent pain also dominate concerns about management in dental settings.[3,15,21–23] Pain is also a subjective response and is influenced by past and present experiences, ethnic and sex differences, and cultural factors.[3,15,21–23] Depression, anxiety, somatic focus, and catastrophizing are human emotions that affect pain, and the interplay of neuropathic trigeminal pain symptoms and behavioral status has not been extensively researched. Little is known about genetic and familial associations to neuropathic trigeminal pain and the degree to which genetic influences provide added risk for or protection from neuropathic trigeminal pain given the same biologic challenge or damage.

Those involved with the dental management of patients presenting with possible neuropathic trigeminal pain are faced with a condition that has highly varied descriptive characteristics between affected patients. For 1 patient, the condition will cause severe allodynia[3,11,21]; another will describe the most debilitating symptom as "burning" or "drawing." Still others describe an urge to "rip out" the teeth or gums. Some avoid touching the region of pain, while others find transient symptom reduction with light tactile or firm pressure sensation in the region of pain.

The region and extent or distribution of the pain can provide further difficulty in assessment. Some patients have extremely focal sites that are extremely sensitive to any stimulation, while others have lower grade pain over wider neurological distributions. In some, the hyperalgesia is so light that it is assumed to be coming from muscle dysfunction.

To gain a better understanding and appreciation for the variation in signs, symptoms, pre-existing states, prior treatments, and neuropathic trigeminal pain treatments of patients seen at our clinic, the cases of 50 consecutive patients presenting to the clinic for follow-up as part of their normal management process were reviewed.

**Table 1a** Demographics and Characteristics of Patient Cases (n = 50)

| | | Range |
|---|---|---|
| Age (y) | 55 (mean) | 40 to 72 |
| Sex | 89% female | |
| Duration of pain | > 5 y (mean) | 1 mo to 26 y |
| Amount of pain per day* | > 12 h (mean) | Seconds to constant |
| Worst pain | 8/10 | 2 to 10 |
| Average pain | 6/10 | 1 to 9 |

*including paroxysmal, episodic, persistent, or escalating pain.

**Table 1b** Pain Location (n = 50)

| Site | Incidence (%) |
|---|---|
| Tooth | 33 |
| Gingiva | 42 |
| TMJ | 16 |
| Trigeminal divisions V2 and/or V3 | 26 |
| Face | 31 |
| Tongue | 15 |
| Other | 10 |

## Case Reviews

### Background

The orofacial pain clinic of the University of Washington is responsible for the diagnosis and management of approximately 7,000 patients per year. These patients account for approximately 10,000 clinic visits for the diagnosis and management of acute or chronic orofacial pain disorders. Approximately 1,500 of the patients are referred to the clinic with complaints of chronic orofacial pain and about 20% of those with chronic pain are ultimately given a diagnosis of chronic neuropathic trigeminal pain. Those provided with that diagnosis exhibit important cardinal symptoms of neuropathic trigeminal pain, including mechanical and/or thermal allodynia, hyperalgesia, or hyperpathia. Once local factors have been ruled out, patients are assigned a final diagnosis of primary neuropathic trigeminal pain if no other specific etiology is determined. Some patients have concurrent secondary pain conditions that are often aggravated by the neuropathic pain, including muscle guarding, muscle tension, jaw posturing, and damaging counterirritation rituals such as repetitive tongue movements.

### Case Features

Of the 50 consecutive patients reviewed, 38% were determined to have neuropathic trigeminal pain, and 62% suffered from TMD and other chronic pain states. The major characteristics of the population are listed in Table 1. The vast majority were females (89%), with pain durations of 1 month to 26 years. Pain severity was widely different, with a low score of 2/10 and a high score of 10/10 reported for the worst pain in the group. Most patients rated their most severe pain as being 8/10. Average pain score also varied widely, with the average pain rated as 6/10. Patients were highly varied in their report of the temporal characteristics of the pain; they reported persistent, episodic, paroxysmal, or escalating pain.

**Table 2** TMD Symptoms, Pain Characteristics, and SCL–90 Status

| | Incidence (%) |
|---|---|
| Symptoms of jaw dysfunction | |
| Clicking | 36 |
| Crepitus | 5 |
| Clenching | 36 |
| Chewing pain | 26 |
| Jaw stiffness | 26 |
| Bite changes | 21 |
| Pain characteristics | |
| Burning | 36 |
| Stabbing | 31 |
| Shooting | 31 |
| Aching | 57 |
| Sickening | 10 |
| SCL–90 | |
| Low | 52 |
| High | 21 |

SCL–90 = Symptom Checklist–90. A low score indicates that the subject is negative for depression/anxiety/somatization.

Table 1 also demonstrates the wide distribution of pain locations, with pain in the tooth, gingiva, face, and TMJ reported by 33%, 42%, 31%, and 16% of the patients, respectively. No clear preference for right or left side was identified. The pain distribution suggests that finding a strict anatomical distribution for chronic orofacial pain may be less common than often suggested, except in cases of clear local nerve injury.

The records of patients with neuropathic trigeminal pain were reviewed to determine comorbid complaints of neuropathic trigeminal pain and TMD (Table 2). Interestingly, symptoms and findings suggestive of TMD were noted in the patients with neuropathic trigeminal pain at the rate expected in the adult population.

Pain descriptors were also reported using the McGill Pain Questionnaire.[23] There was a high degree of variation in choice of words. Only 10% described the pain as sickening, whereas more than

**Table 3** Factors Associated with Onset and Prior Treatment Reported by the Patients

| | Incidence (%) |
|---|---|
| Associated onset factors | |
| Trauma | 16 |
| Implant placement surgery | 31 |
| Dental injection | 10 |
| Spontaneous | 26 |
| Clenching | 15 |
| Prior treatments | |
| Endodontics | 36 |
| Implant removal | 21 |
| Restorative | 26 |
| Splint and TMD treatment | 42 |
| Surgery | 21 |

**Table 4** Medications and Other Treatment Approaches

| | Incidence (%) |
|---|---|
| Treatment medications | |
| Topical medications | 42 |
| Gabapentin | 31 |
| Carbamazepine | 16 |
| Antidepressants | 75 |
| Topiramate or Depakote | 10 |
| Opioid analgesics | 78 |
| Dental treatments | |
| Topical medications | 42 |
| Removal of pressure on tissue | 21 |
| Protective splints | 30 |
| Local ablation | 10 |
| Topical anesthetics | 30 |

50% used the less behaviorally loaded word "aching." Interestingly, burning was selected more often than words more commonly thought to describe neuropathic orofacial pain (shooting and stabbing), although the latter terms were used by almost a third of the subjects (Table 2).

The Symptom Checklist-90 (SCL-90) was used to assess behavioral status.[24] The rate of high SCL scores for depression, anxiety, and somatic focus approximated that seen in studies of other chronic facial pains, such as TMD (Table 2).[15]

As part of routine case evaluation, patients were also asked to identify the factors that they thought were associated with the onset of the complaint (Table 3) and prior treatments that had been tried to control or to eliminate the condition (Table 3). Surgery associated with dental implants was the most commonly reported factor, although it can be speculated that some patients may have been confused and attributed the onset to the surgery when the site had pre-existing pain that led to an earlier dental extraction. More than 25% could identify no initiating factors and described the onset as spontaneous and without provocation. More than 40% of the patients had received splint therapy for TMD pain, but without success, and more than a third had received endodontic therapy. None of the interventions provided relief, and a number of patients reported that the dental interventions tended to increase symptoms and pain scores.

Prescribed medications directed at symptom control are outlined in Table 4. Topical anesthetics and topical antidepressants with anesthetic properties were commonly used for short-term pain control. Traditional antineuralgia and neuropathic pain medications as well as narcotic analgesics were also commonly employed. Results were mixed; some patients reported modest symptom relief, while others discontinued the medication because of side effects or lack of benefits. Table 4 also identifies other treatment approaches used with reasonable outcomes. Daily topical application of anesthetics, topical steroids, and antidepressants were most commonly used and significantly reduced pain scores.

## Case Implications

The number of subjects reporting spontaneous onset of their condition was high, which suggests that if trauma is a key factor in the initiation of neuropathic trigeminal pain, then the amount and extent of trauma may vary considerably. Pain may start as a result of events and procedures that may seem relatively innocuous, such as oral trauma associated with events of daily living or dental procedures such as anesthetic infiltration or dental prophylaxis. As might be expected, invasive dental procedures, including extractions and surgery, were often associated causally with the initiation of the problem. The significant percentage of patients who identified the gingiva as the site of pain was unexpected and may deserve additional exploration, since pain in the gingiva and around the teeth frequently leads to additional invasive dental procedures including endodontic therapy, extraction, restorative care, and occlusal adjustment. The high pain scores were expected but the atypical presentation of neuropathic trigeminal pain was not expected. While a reasonable percent of patients exhibited shooting and paroxysmal symptoms, which are typical of neuropathic trigeminal pain, aching and burning sensations, which are not symptoms normally thought to accompany classical neuropathic trigeminal pain, were even more common.

The high prevalence of concomitant TMD symptoms was also surprising, suggesting either that some forms of neuropathic trigeminal pain trigger secondary TMD or that both sensory and motor changes exist in some varieties of neuropathic trigeminal pain.[13,14] The author's experience with neuropathic trigeminal pain has been that motor involvement can occur, and some patients exhibit significant motor changes equivalent to that seen in focal dystonias and myoclonic contractions.[18] The data clearly demonstrate that no single treatment is effective and that topical approaches may provide some level of short to mid-term symptom reduction.

## Conclusions

Correct diagnosis, and particularly early definitive diagnosis, of neuropathic trigeminal pain is crucial to avoid invasive and potentially more damaging forms of treatment. Many clinicians may not recognize symptoms of neuropathic trigeminal pain and often diagnose the presentation of it as consistent with TMD and/or dental pathology. The overlap of symptom characteristics between some variants of neuropathic trigeminal pain and classic dental disorders may be responsible for this confusion. Primary care physicians may also lack the training to establish a diagnosis of neuropathic trigeminal pain. More thorough examination of periodontal, alveolar, and gingival tissues for regions of allodynia, hyperpathia, and hyperalgesia will improve detection of less severe and more atypical cases of neuropathic trigeminal pain. The use of topical anesthetic agents may be of value in initial assessment and also in the diagnosis of neuropathic trigeminal pain that presents as surface mucosal hyperpathia and hyperalgesia. However, specific medications do not appear to be dramatically effective across all types of neuropathic trigeminal pain, and some of the responses to medications may relate to a general effect and an effect on behavioral status (eg, placebo response) more than correction of specific pathophysiologic defects in nerve conduction or transmission. It is also important to recognize that pain conditions exist along at least a 2-axis parameter—a behavioral or psychological axis and a biophysical axis. Addressing issues along the behavioral axis can often provide significant symptom reduction even though the basic neuropathologic condition persists.

To improve our understanding of neuropathic trigeminal pain, population-based epidemiologic studies of incidence and prevalence along with other factors are essential. Diagnostic criteria need to be established, tested, and refined. Educational initiatives to improve our understanding of neuropathic trigeminal pain and thus its assessment and diagnosis are vital to the establishment of standardized treatment protocols. Long-term success in prevention, diagnosis, and management of neuropathic orofacial pain will also be determined by progress in research that focuses on the basic and molecular mechanisms of pain and behavior.

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