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Charles S. Greene and Greg M. Murray

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Atypical odontalgia

An oral neuropathic pain phenomenon

Charles S. Greene, DDS; Greg M. Murray, MDS, PhD

CLINICAL PROBLEM

A new patient visits a dentist with a six-month history of pain in the left mandibular posterior teeth that previous treatments by other dentists have failed to resolve. She is relatively caries free and has only occlusal restorations in a few teeth. The pain was first diagnosed by her previous general dentist as pulpitis in the left mandibular permanent first molar, but neither pulpectomy nor endodontic treatment relieved the pain. The patient was convinced that the pain was in an adjacent tooth, so her dentist agreed to explore that tooth, leading to more unsuccessful endodontic treatment. An endodontist re-treated both teeth and performed apical surgery on one of them, but no improvement occurred. Now the patient is convinced that the pain is emanating from other teeth in the region, and she is angry with her previous dentists for not resolving this problem.

EXPLANATION

It is likely that this phenomenon is an example of a certain type of neuropathic pain that in the past has been labeled with such names as "phantom tooth pain" and "atypical facial pain." However, in recent years this phenomenon most widely has been called "atypical odontalgia" (AO) (a discussion of new terminology appears below). To provide better understanding of this, we present a brief review of neuropathic pain, followed by a discussion of AO, with special emphasis on how the treatment of patients with such pain should be managed in the dental office. Early recognition of this diagnosis is the key to avoiding the scenario in the case described here, and later encounters require a careful approach to avoid further trouble.

Overview of neuropathic pain. A noxious stimulus to any peripheral structure activates nociceptors or pain receptors, producing nerve impulses that travel to the brain for the perception of pain. This acute pain often is termed "nociceptive pain" because it arises from the direct activation of nociceptors. Neuropathic pain, on the other hand, is pain arising as a result of a lesion or disease affecting the actual nerves that convey touch, pressure, pain and temperature information from the body (skin, muscles, joints, bone and so forth) to the brain.[1] In some cases, neuropathic pain can arise completely spontaneously, for reasons that are not yet understood.

The figure shows one possible neural mechanism involved in the clinical case described above, a mechanism that other authors also have described.[2-4] An initial pulpitis or endodontic treatment in the left mandibular molar can damage the pain nerves near the apex of this tooth (labeled A in the figure). This type of nerve injury usually heals uneventfully after the patient undergoes endodontic procedures and extractions, and there is no residual pain, but occasionally such injuries can initiate a deafferentation response in which the apical nerves become hypersensitive. In some patients, for unknown reasons (possibly genetic factors), a nerve tissue structure called a "neuroma" develops. The pain nerve fibers in these neuromas become sensitized by activated glial and immune cells within the neuroma. Nerve impulses can fire off spontaneously, and the patient may experience spontaneous pain even though all the original infection has been

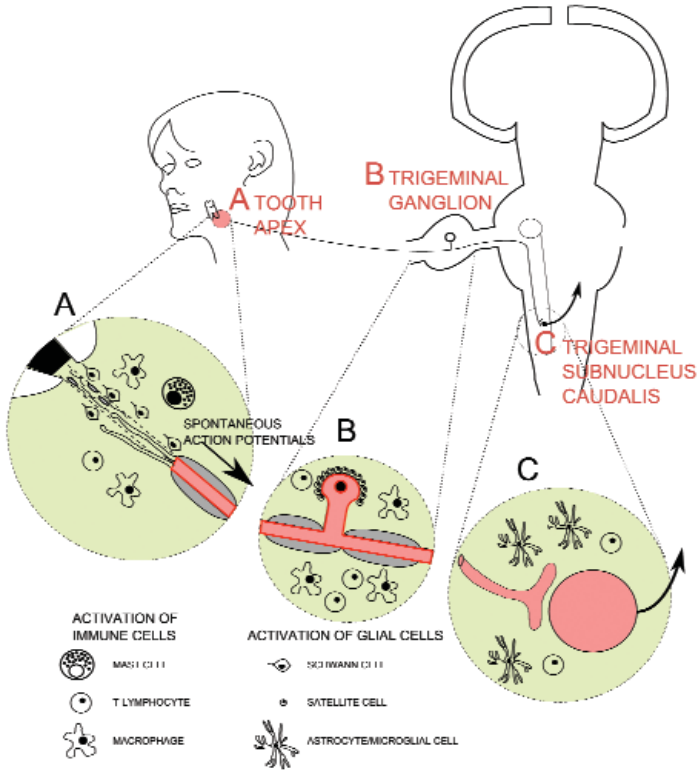

Figure. Diagram showing how neuropathic pain arises and is transmitted to the central nervous system.

removed and the area has "healed." The trigeminal ganglion (B in the figure) and the trigeminal subnucleus caudalis (C in the figure), which relay this pain information, also can become sensitized by activated immune cells and glial cells within those structures, and this can enhance the spontaneous pain further.

Therefore, in the absence of any identifiable peripheral lesion in these susceptible people, an abnormal spontaneous activity in these nerves may continue to occur, which will result in the person's experiencing persistent pain. For reasons that were described in previous articles,[5,6] the symptoms of toothachelike pain in other teeth are likely to be a manifestation of referred pain, which involves neuroplastic changes in the brain.

Atypical odontalgia: changing terminology. The term "atypical odontalgia" is being revised by the Orofacial Pain Special Interest Group of the International Association for the Study of Pain. The new term, which is much more specific, will be "chronic continuous dentoalveolar pain" (CCDAP). The diagnostic criteria for this condition are

- chronic, continuous pain;
- pain localized in the dentoalveolar region;
- pain not caused by another disorder.

This change from AO to CCDAP is part of a major overhaul of terminology in the orofacial pain field that should lead to an improved diagnostic taxonomy.

OUTCOME, CONCLUSIONS AND CLINICAL IMPLICATIONS

The clinical case we report here was resolved after the dentist sent the patient for appropriate medical management of her chronic neuropathic pain. Such management can be accomplished by orofacial pain and oral medicine experts who have had formal postdoctoral training, as well as by neurologists and other pain management physicians. The main message for dentists is the need to be cautious about getting drawn into performing multiple dental procedures when these situations arise. The patients often are convinced that their pain is localized in a tooth or teeth—and convincing in stating so—but in most cases the dental findings simply do not support that assumption. Also, it is important to remember that repeated operations in the painful region not only will fail to produce relief, but also, in many cases, the pain gets worse with each intervention. Patients who appear to have this kind of problem should be told that they need to see an orofacial pain expert—preferably sooner rather than later. If the diagnosis proves to be neuropathic pain, both the patient and the dentist will be glad that unnecessary and expensive dental treatment was avoided. ■

Dr. Greene is a clinical professor, Department of Orthodontics, College of Dentistry, University of Illinois at Chicago. He is also the section editor for Pain Update. Address reprint requests to Dr. Greene at Department of Orthodontics (M/C 841), College of Dentistry, University of Illinois at Chicago, 801 S. Paulina, Room 131, Chicago, Ill. 60612-7211, e-mail "cgreene@uic.edu".

Dr. Murray is a professor, Jaw Function and Orofacial Pain Research Unit, Faculty of Dentistry, University of Sydney, Centre for Oral Health, Westmead Hospital, New South Wales, Australia.

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1. Treede RD, Jensen TS, Campbell JN, et al. Neuropathic pain: redefinition and a grading system for clinical and research purposes. Neurology 2008;70(18):1630-1635.
2. Marchand F, Perretti M, McMahon SB. Role of the immune system in chronic pain. Nat Rev Neurosci 2005;6(7):521-532.
3. Scholz J, Woolf CJ. The neuropathic pain triad: neurons, immune cells and glia. Nat Neurosci 2007;10(11):1361-1368.
4. Woda A, Salter MW. Mechanisms of neuropathic pain. In: Sessle BJ, Lavigne GJ, Lund JP, Dubner R, eds. Orofacial Pain: From Basic Science to Clinical Management. 2nd ed. Hanover Park, Ill.: Quintessence; 2008:53-59.
5. Greene CS. Neuroplasticity and sensitization. JADA 2009; 140(6):676-678.
6. Murray GM. Referred pain, allodynia and hyperalgesia. JADA 2009;140(9):1122-1124.