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Dental treatment for patients with neuropathic orofacial pain

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CLINICAL PROBLEM

A 56-year-old woman visited a dental office with acute dental pain in the left mandibular molar area. She had not received routine dental care during the past several years. Approximately three years previously, she experienced multiple daily episodes of spontaneous pain, induced pain or both on the left side of her face following the distribution of the left mandibular nerve (V3 branch). This pain was intermittent, severe, sharp, electriclike and lancinating, with bursts of short duration (that is, lasting seconds to one minute). The patient reported avoiding any form of stimulation to this area as this could trigger these excruciating episodes. The dentist (G.D.K.) obtained a comprehensive history and performed an examination with the aid of appropriate imaging to rule out central nervous system pathology. He established a diagnosis of trigeminal neuralgia (TN) related to the left V3 branch, for which the patient was treated with nerve membrane-stabilizing anticonvulsants. To date, the medication controls the TN and the patient experiences only two to three brief, but painful, episodes (spontaneous, induced or both) on a weekly basis.

The patient's medical history included mild hypertension, hypothyroidism and intermittent low back pain for which she was being treated adequately by her physician. The clinical examination revealed no problems with her intraoral soft tissues. The left mandibular permanent first molar (tooth no. 19) had a large defective restoration that responded equivocally to vitality testing and thermal challenge (cold), with

no percussion sensitivity and no evidence of radicular pathology. The dentist determined that placement of a full metal crown would be the treatment of choice for the restoration of tooth no. 19.

The dentist now faces a dilemma related to provision of necessary dental care in a region of current neuropathic orofacial pain (NOP). The intervention to be provided poses the potential risk of reactivating the initial pain or of exacerbating the patient's ongoing condition. The dentist will need to modify dental procedures to minimize potential adverse consequences in these unusual circumstances.

EXPLANATION

The International Association for the Study of Pain defined neuropathic pain as "pain caused by a lesion or disease of the somatosensory nervous system."¹ However, when a patient experiences neuropathic pain, he or she may have no demonstrable lesion or disease. This discrepancy between the definition and clinical manifestation may be explained by the work of Costigan and colleagues,² who referred to this as "dysfunctional pain." Dysfunctional pain is considered to represent a malfunction (which can be considered a disease unto itself) of the somatosensory nervous system, involving both spontaneous and stimulus-dependent pain (evoked by both low- and high-intensity stimuli) but without known structural nervous system lesions or active peripheral inflammation (for discussions pertaining to NOP, see Pain Update articles about neuroplasticity³ and atypical odontalgia⁴).

For clinical utility, NOP may be categorized as continuous or episodic. The patient in this case originally had episodic NOP (that is, intermittent periods of pain) that consisted of classical signs and symptoms consistent with TN.⁵ The category of NOP that is continuous is characterized by constant, ongoing and unremitting pain, with fluctuating intensities of pain. Often, TN is associated with a perioral trigger zone, an intraoral trigger zone or both, whereby nontraumatic stimuli such as a light touch provoke severe paroxysmal pain.⁵ Because of this, patients with current or controlled TN may be reluctant to participate in any activity or undergo any dental procedure (preventive or restorative) that involves manipulation to these hypersensitive areas.

CLINICAL IMPLICATIONS

Patients experiencing NOP inevitably will require dental treatment on an acute basis or for the routine maintenance of oral health. However, owing to the fear of reigniting the original pain or amplifying their current pain, they may avoid these procedures. Unfortunately, this avoidance may lead to progressive dental disease, with the additional consequence of barrages of nociceptive input into an existing dysfunctional nervous system. Effective oral health maintenance may reduce the need for invasive dental treatments, with the added benefit of reducing the patient's risk of developing complications associated with NOP. When invasive procedures are required, it is of utmost importance for the dental team to recognize and understand these issues and to select the least invasive approaches to treatment.^{6,7} Although the case described here is of a patient with TN (episodic NOP), the treatment approaches discussed below are equally applicable to NOP that is continuous.

Patient scheduling. The goal of every practitioner should be to provide maximum comfort during and after the procedure. The intensity of NOP has a tendency to fluctuate throughout the day; therefore, dental appointments may best be planned during periods of lowest pain intensity or during periods of remission (as is possible with TN). Clinicians should perform dental procedures when medication used in the treatment of these conditions is at its peak level of effectiveness, which requires a thorough understanding of the characteristics of the medication or medications being used to manage the patient's pain.

Local anesthetic and preemptive analgesia. The administration of local anesthetic and

use of needles, although performed on a daily basis and rarely associated with complications, may be a cause of pain exacerbation in patients with NOP.^{8,9} Furthermore, possible neurotoxicity associated with the local anesthetic may have an effect on the patient.^{8,10-12} Other factors to be considered with regard to the choice of local anesthetic are related to its concentration and duration of exposure to peripheral neural tissue.¹³ To minimize the risk of the patient's experiencing an increase in pain, the clinician should administer the local anesthetic carefully, avoiding undue tissue trauma and using agents that have minimal neurotoxic and ischemic potential, with minimal concentrations and exposure times. He or she also should consider administering booster doses of an antineuropathic medication or medications before invasive procedures.¹⁴ In addition, the clinician should administer a long-acting anesthetic at the end of the procedure to delay postoperative discomfort.^{15,16} When procedures are performed under general anesthesia, the addition of a local anesthetic will reduce peripheral and central sensitization.^{14,17,18} Behavioral medicine approaches, such as hypnosis, relaxation techniques or both, may support pain control or pain suppression.¹⁹

Preventive and hygiene procedures.

To minimize stimulation, which can lead to an increase in the patient's NOP, as well as to prevent the patient from avoiding normal daily dental hygiene, the dental team can modify dental procedures and techniques for plaque control. Atraumatic oral hygiene may be facilitated by using an ultrasoft toothbrush or a soft foam brush soaked in chlorhexidine.²⁰ Interdental cleaning may be facilitated by use of an assortment of devices, such as floss of various textures, dental tape and interdental cleaners. Clinicians should consider the use of antiseptic or antiplaque alcohol-free mouthrinses, recalcifying and remineralizing agents, and fluoride supplementation (in high-risk patients) delivered in soft custom trays. They should perform scaling and prophylaxis with care and may need to administer local anesthetic to the areas that will be treated to decrease nociception.

Least invasive approach. Practitioners should consider the least invasive approach to treatment. As discussed earlier, prevention is the first choice. The following are examples of approaches that may pose a lower risk to the patient with regard to aggravation of pain:

- crown amputation and retention of roots rather than extraction;
- site selection for implants outside of the immediate zone of NOP;

■ use of a removable prosthesis rather than a fixed prosthetic device, which also can serve as a vehicle for application of transmucosal medications.

CONCLUSIONS

Dentists need to recognize and understand the concepts of NOP to provide appropriate treatment for patients. To provide the highest-quality dental care for this unique patient population, they must incorporate into the final treatment plan factors such as communication among all health care practitioners and the patient, appreciation of and respect for the patient who is experiencing NOP and understanding the patient's tolerance for potentially painful procedures. ■

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1. International Association for the Study of Pain (IASP). IASP taxonomy. www.iasp-pain.org/Content/NavigationMenu/GeneralResourceLinks/PainDefinitions/default.htm#Neuropathicpain. Accessed July 15, 2013.

2. Costigan M, Scholz J, Woolf CJ. Neuropathic pain: a maladaptive response of the nervous system to damage. *Annu Rev Neurosci* 2009;32:1-32.

3. Greene CS. Neuroplasticity and sensitization. *JADA* 2009;

140(6):676-678.

4. Greene CS, Murray GM. Atypical odontalgia: an oral neuropathic pain phenomenon. *JADA* 2011;142(9):1031-1032.

5. Scrivani SJ, Mathews ES, Maciewicz RJ. Trigeminal neuralgia. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2005;100(5):527-538.

6. Remick RA, Blasberg B, Barton JS, Campos PE, Miles JE. Ineffective dental and surgical treatment associated with atypical facial pain. *Oral Surg Oral Med Oral Pathol* 1983;55(4):355-358.

7. Mock D, Frydman W, Gordon AS. Atypical facial pain: a retrospective study. *Oral Surg Oral Med Oral Pathol* 1985;59(5):472-474.

8. Hillerup S, Jensen RH, Ersboll BK. Trigeminal nerve injury associated with injection of local anesthetics: needle lesion or neurotoxicity? *JADA* 2011;142(5):531-539.

9. Pogrel MA, Thamby S. Permanent nerve involvement resulting from inferior alveolar nerve blocks (published correction appears in *JADA* 2000;131(10):1418). *JADA* 2000;131(7):901-907.

10. Haas DA, Lennon D. A 21 year retrospective study of reports of paresthesia following local anesthetic administration. *J Can Dent Assoc* 1995;61(4):319-330.

11. Kasaba T, Onizuka S, Takasaki M. Procaine and mepivacaine have less toxicity in vitro than other clinically used local anesthetics. *Anesth Analg* 2003;97(1):85-90.

12. Smith MH, Lung KE. Nerve injuries after dental injection: a review of the literature. *J Can Dent Assoc* 2006;72(6):559-564.

13. Selander D. Neurotoxicity of local anesthetics: animal data. *Reg Anesth* 1993;18(6 suppl):461-468.

14. Foreman PA. Preemptive analgesia: the prevention of neurogenic orofacial pain. *Anesth Prog* 1995;42(2):36-40.

15. Gordon SM, Dionne RA, Brahim J, Jabir F, Dubner R. Blockade of peripheral neuronal barrage reduces postoperative pain. *Pain* 1997;70(2-3):209-215.

16. Gordon SM, Mischenko AV, Dionne RA. Long-acting local anesthetics and perioperative pain management. *Dent Clin North Am* 2010;54(4):611-620.

17. Kaufman E, Epstein JB, Gorsky M, Jackson DL, Kadari A. Preemptive analgesia and local anesthesia as a supplement to general anesthesia: a review. *Anesth Prog* 2005;52(1):29-38.

18. Woolf CJ, Chong MS. Preemptive analgesia: treating postoperative pain by preventing the establishment of central sensitization. *Anesth Analg* 1993;77(2):362-379.

19. Carlson CR. Psychological considerations for chronic orofacial pain. *Oral Maxillofac Surg Clin North Am* 2008;20(2):185-195.

20. Ransier A, Epstein JB, Lunn R, Spinelli J. A combined analysis of a toothbrush, foam brush, and a chlorhexidine-soaked foam brush in maintaining oral hygiene. *Cancer Nurs* 1995;18(5):393-396.