Oral local anesthesia successfully ameliorated neuropathic pain in an upper limb, suggesting pain alleviation through neural plasticity within the central nervous system: a case report

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Purpose

Neuropathic pain typically appears following peripheral nerve injury in neuropathies, plexopathies, and trauma to selected sites within the central nervous system (CNS). Recently, evidence-based recommendations for pharmacological treatments of neuropathic pain have been proposed, on the basis of both positive and negative results from multiple randomized controlled trials. However, approximately 10% to 15% of all neuropathic pain patients are refractory to pharmacotherapy. For these cases, more invasive pain-management interventions, such as intrathecal drug delivery, neurostimulation, or neural blockade, may be used. Ideally, blocking neural transmission, either temporarily by using local anesthetics or permanently by surgical nerve ablation, could reduce neuropathic pain; however, no neural blockades have been found to be consistently successful. Here, we report a case of a patient with postbrachial plexus avulsion injury pain whose neuropathic pain had been refractory to several evidence-based pharmacotherapies such as spinal cord stimulation and epidural and brachial plexus blockades. His pain could be well-controlled by oral local anesthesia, suggesting pain alleviation through neural plasticity within the CNS.

Method

A 49-year-old man suffered a complete left brachial plexus avulsion injury 10 years ago and experienced severe neuropathic pain in his left upper limb immediately after the trauma. He had been treated several times for pain through a left brachial plexus blockade and a cervical epidural blockade, with no success. His neuropathic pain decreased slightly upon prescription of pregabalin and application of cervical spinal cord stimulation (SCS), but it still remained severe. He once underwent a dental treatment for his left mandibular molar tooth. When local anesthesia was applied near the left mandibular molar tooth (3 ml, 0.5% lidocaine), he felt the enlargement of that region, following which his neuropathic pain disappeared immediately. Approximately two hours after the dental treatment, the neuropathic pain returned and gradually increased to predental treatment levels. A nonsteroidal anti-inflammatory drug, loxoprofen, completely ameliorated the dental pain but was not effective against neuropathic pain. Since then, the patient had 3 dental treatments, and local anesthesia on the left molar tooth consistently ameliorated neuropathic pain. Analgesic effects consistently lasted for several hours following the administration of local anesthesia. His neuropathic pain could be mildly controlled by a combination of pregabalin, SCS, and local anesthesia near the left molar tooth, although the molar tooth has completely improved. The use of oral local anesthesia for breakthrough neuropathic pain has been especially useful.

We obtained the patient's consent to report his progress, in accordance with the Declaration of Helsinki.

Results

Under conditions of neuropathic pain, particularly in deafferentation pain following massive nerve injury such as postamputation phantom limb pain; postbrachial plexus injury pain; and postspinal cord injury pain, cerebral somatotopic reorganization in the sensorimotor cortices of the brain is observed. Following deafferentation of an upper limb by nerve injury, the somatotopic region responding to the upper limb in the sensorimotor cortices shrinks
and instead the somatotopic region responding to the facial region, which is located next to the upper limb, expands. The degree of shrinkage of the upper limb representation correlates linearly with severity of neuropathic pain. Further, expansion of the somatotopic representation of the affected body part correlates with pain alleviation through neurorehabilitation techniques. Therefore, somatotopic reorganization in the sensorimotor cortices closely relates to pathophysiological mechanisms underlying neuropathic pain and its alleviation.

We consider that analgesic effects of the oral local anesthesia in our case were derived from such neural plasticity in the sensorimotor cortices. Deafferentation by local anesthesia, as well as that by nerve injury, shrinks the somatotopic representation of the exposed body part and simultaneously expands a nearby somatotopic representation in the sensorimotor cortices. On the basis of this information, we speculated that, in our case, local anesthesia in the mouth could have shrunk the mouth/face representation and subsequently expanded somatotopic representation of the hand/upper limb within the sensorimotor cortices, resulting in the amelioration of neuropathic pain in the upper limb. Local anesthesia at an intact limb contralateral to the painful limb has been reported to display clear analgesic effects on postamputation phantom limb pain, suggesting pain alleviation through neural plasticity within the CNS. Analgesic effects on neuropathic pain from local anesthesia are derived from CNS plasticity.

**Conclusions**

For neural blockades, oral local anesthesia is a novel candidate for treating neuropathic pain in the upper limb, and the analgesic effect might be derived from its effects on neural plasticity within the CNS.