Injection of botulinum toxin type A (BOTOX) into trigger zone of trigeminal neuralgia as a means to control pain

Wei Cheong Ngeow, BDS (Mal), FFDRC (Ireland), FDSRCS (Eng), MDSc (Mal), a and
Rekha Nair, MBBS (India), DRM (Mal), LFOMRCPI (Ireland)b

This article illustrates a case of persistent trigeminal neuralgia in a medically compromised 65-year-old female who did not respond to pharmacotherapy. She had undergone several peripheral neurectomies as well as a failed right posterior fossa exploration that resulted in a cerebrospinal fluid leak. Persistent pain over the right external nasal area and right mental region was relieved for several hours after daily injections of bupivacaine. A trial of a single dose of 100 units of botulinum toxin type A (BOTOX) diluted in 2.5 mL saline was injected into the external nasal trigger zone (60 units) and into the mental nerve region (40 units). She achieved complete pain relief in the external nasal region for 5 months. Pain recurred and the site was again injected with 100 units of botulinum toxin type A (BOTOX). Pain relief at the mental region was partial. This was finally controlled with peripheral neurectomy. The patient was pain free with a maintenance dose of 200 mg carbamazepine daily for about 1 year, after which she elected to undergo stereotactic gamma knife radiosurgery when pain recurred at the external nasal region. (Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2010;109:e47-e50)

Trigeminal neuralgia (TN) is a severe chronic pain syndrome characterized by an excruciating, brief electric shocklike paroxysmal pain in one or more divisions of the trigeminal nerve. It can occur either spontaneously or upon gentle tactile stimulation of a trigger zone on the face or in the oral cavity.1-5 Various theories have been proposed on its etiology, among which is that the painful paroxysms result from epileptic seizures in the brain stem trigeminal structures.6 Other probable causes are trigeminal root compression by adjacent arterial loops, or occasionally by tumors, cysts, arteriovenous malformations, or aneurysms.6 An earlier theory suggested a peripheral etiology.4

About 7 years ago, the ignition hypothesis was forwarded to explain the pathophysiology of TN.3 According to this hypothesis, TN results from specific abnormalities of trigeminal afferent neurons in the trigeminal root or ganglion. Injury leaves axons and axotomized somata hyperexcitable. The hyperexcitable afferents give rise to pain paroxysms as a result of synchronized after discharge activity. The ignition hypothesis accounts for the pathogenesis of TN, for its major clinical signs, and for the efficacy of treatment modalities.3

There are 2 major means of treatment for TN, namely pharmacotherapy and surgical procedures.7 Medical management is the mainstay treatment for trigeminal neuralgia.1,5 Some of the more successful pharmacotherapy means include the use of antiepileptic drugs like carbamazepine, with secondary drug choices being baclofen, lamotrigine, oxcarbazepine, phenytoin, gabapentin, and sodium valproate.5 Pharmacotherapy generally carries less risk than major surgical procedures and is suitable for medically compromised patients who are unfit for such surgery.

Surgical treatment may be an alternative for patients who do not respond well to medical management or are severely affected by their side effects.1 There are essentially 2 targets for surgical approaches: the peripheral nerve and the trigeminal ganglion. Microvascular decompression (MVD), which targets the trigeminal ganglion, has been reported to be the most successful neurosurgical procedure in treating TN.5,7 Other central procedures include radiofrequency neurolysis, balloon compression procedures, and stereotactic gamma knife radiosurgery. Peripheral neurectomy, cryotherapy, and peripheral nerve injection with glycerol/ethanol or streptomyacin are some other options that produce temporary relief and are useful for patients who are unfit to undergo major neurosurgery.8

In the past few years, there have been several reports on the successful use of botulinum toxin type A as an agent for peripheral nerve injection in patients with TN.6,12 However, 2 of the cases reported were not in patients with typical TN.9,10 In the only open-label