

Supraorbital Nerve Electric Stimulation for the Treatment of Intractable Chronic Cluster Headache: A Case Report

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We describe a patient with intractable chronic cluster headache that responded well to supraorbital nerve electric stimulation.

Key words: cluster headache, supraorbital nerve neuromodulation, ophthalmic nerve

Cluster headache is a primary neurovascular headache. It is strictly unilateral head pain mainly in the ophthalmic distribution of the trigeminal nerve that is associated with cranial autonomic symptoms and usually follows a circadian and circannual pattern.

There are 2 forms of cluster headache, episodic and chronic. Episodic cluster headache occurs in periods from 7 days to 1 year separated by pain-free periods lasting at least 1 month. Chronic cluster headache occurs over the interval of more than 1 year without remission or with remission lasting less than 1 month.^{1,2}

Chronic cluster headache accounts for about 10% of patients with cluster headache and it usually lacks the circadian pattern typical of the episodic one. Patients with chronic cluster headache are often resistant to pharmacological management as they are more prone to tachyphylaxis and eventual loss of response.² Here we report, for the first time, the effectiveness of supraorbital nerve neuromodulation in the management of a patient with medically intractable chronic cluster headaches.

CASE REPORT

We report on a 35-year-old male with episodic cluster headache for the last 5 years. His headaches lost circadian pattern and occurred continuously without remissions for the last 2 years. He continued to complain of 2–3 attacks of classic cluster headaches per day, each lasting for 1 1/2 to 2 hours. His attacks do consist of excruciating right orbital and

supraorbital pain associated with lacrimation, conjunctival injection, rhinorrhea, eyelid edema, and ptosis. The patient became resistant to subcutaneous sumatriptan, parenteral dihydroergotamine (DHE), nasal oxygen, and narcotics. He failed multiple preventive pharmacological therapies including verapamil (840 mg/day), lithium (1200 mg/day), valproic acid (1600 mg/day), neurontin (3600 mg/day), topamax (300 mg/day), as well as 2 courses of oral steroids (prednisolone 60 mg/day). Greater occipital nerve block and sphenopalatine ganglion block provided only short-term relief. Following appropriate psychological evaluation and evaluation by our institutional committee for implantable devices, he was felt to be a good candidate for a trial of right supraorbital nerve stimulation. He underwent a successful 7-day trial with a percutaneous electrode (Octrode, ANS Inc., Plano, TX, USA) that was placed horizontally across the course of the supraorbital nerve just above the supraorbital ridge (see Figure). Subsequently, he underwent a permanent implant of the lead that was tunneled and anchored behind the ear and then connected to a rechargeable generator (Eon, ANS Inc.) placed in the infraclavicular area. After the implant, the patient reported good coverage over the supraorbital area.

The patient was provided with 2 different programs. One program for continuous stimulation (1–2 – 3–4 – 5+, 0.8–2 V, 60 Hz, 200 μ s) and the second one to use as a rescue to control the cluster attack, and he was able to abort the attack in few minutes by switching to the second program (2–4+, 2–4 V, 100 Hz, 200 μ s). In addition, his headaches decreased in frequency to one attack every other day. His cluster headaches lasted for only 2 months after the implant and he is in remission over the last 14 months. At the time of the implant, the patient was on verapamil (760 mg/day) and methadone (40 mg/day) and currently he is off both.

As the patient was doing great without headaches 5 months after the implant, he thought that he may be in a spontaneous remission and he turned off the stimulation to

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Figure.—AP view showing right supraorbital percutaneous lead.

save the battery and in 24 hours he developed a severe attack of cluster headache that he was able to abort with turning on the stimulation again.

COMMENTS

The pathophysiology of cluster headache involves trigeminal nociceptive activation (mainly ophthalmic division) with resulting reflex cranial parasympathetic autonomic activation.²

Our proposed theory is that the neuromodulation of afferent input in the ophthalmic division of the trigeminal nerve through electric stimulation of the supraorbital nerve, the terminal cutaneous branch of the ophthalmic nerve, could interfere with the trigeminal-autonomic reflex and abort the attack of cluster headache. Also continuous stimulation of the supraorbital nerve may set the stage for the development of neuromodulation at higher centers involved in the pathogenesis of cluster headaches.

As the headache returned when the stimulator was turned off and it was successfully controlled when it was turned on again, it is more likely that the remission is—at least in part—related to the stimulation and not to the natural history of the disease. However, it is difficult to come up with any conclusion at this point as this will require further studying.

Peripheral nerve stimulation has been used for years in the treatment of intractable neuropathic pain from pe-

ripheral nerve injury. There is growing interest in applying this modality of treatment in the management of occipital neuralgia, migraine headache, and craniofacial pain.³⁻⁷ Positron emission tomography (PET) scan studies showed increased regional cerebral blood flow in areas involved in central neuromodulation in chronic migraine patients with occipital nerve electrical stimulation.⁸

Recently, 2 studies reported the effectiveness of occipital nerve stimulation in the treatment of drug-resistant chronic cluster headaches.^{9,10}

However, to our knowledge, this is the first report to describe the effectiveness of supraorbital nerve stimulation in the management of cluster headaches. This is a relatively simple, safe, minimally invasive, and reversible modality of treatment that may be effective in the management of otherwise one of the most devastating headache that one can experience. It is less invasive and could be safer than hypothalamic deep brain stimulation or other destructive surgical intervention targeting the trigeminal nerve or the cranial parasympathetic outflow.⁹⁻¹²

Conflict of Interest: None

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