Posttraumatic Headache: Surgical Management of Supraorbital Neuralgia

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Background: Supraorbital neuralgia is a distinct clinical entity that presents with episodic, often unilateral, long-lasting attacks of moderate to severe frontal pain. This may ensue following a traumatic or surgical insult to the supraorbital or supratrochlear nerve. Surgical management of these patients is only sporadically discussed in the available literature.

Methods: The authors report a series of six consecutive patients undergoing surgical excision of the supraorbital and supratrochlear nerves on the affected side for refractory posttraumatic or postoperative supraorbital neuralgia. End-to-end nerve coaptation by means of a neural tube conduit was used to prevent future neuroma formation. Success was defined as a 50 percent or greater reduction of preoperative pain level.

Results: Five of six patients demonstrated at least a 50 percent reduction in pain. Three patients experienced complete pain cessation postoperatively. There was one treatment failure. Pain was measured using a visual analogue pain scale. Preoperative average pain was 9.16 ± 1.3 and postoperative average pain was 1.5 ± 1.9, an improvement of 7.7 points or 84 percent ($p = 0.03$). Mean age of the patients was 42 years. Mean follow-up was 14 months. No surgical complications occurred.

Conclusion: Excision of the supraorbital and supratrochlear nerves with end-to-end coaptation of the proximal nerve stumps by means of a neural tube appears to be an effective treatment in selected patients with chronic, posttraumatic supraorbital neuralgia. (*Plast. Reconstr. Surg.* 121: 1943, 2008.)

Chronic posttraumatic or postsurgical headache is a problem that can cause significant morbidity. It has been estimated that 60 percent of patients with this infirmity will require some form of financial aid or support from society. This translates into a high socioeconomic cost in terms of missed work, disability benefits, and early retirement.[1] The intangible burden of suffering on the patient can be devastating as well, causing relationship disturbances, emotional lability, and even suicidal ideation.

A small subset of patients with chronic posttraumatic or postsurgical headache is suffering from supraorbital neuralgia. This is a relatively new and sparsely documented cause for chronic headache. The differential diagnosis of this entity may be somewhat challenging, as it shares features with migraine headache, cluster headache, trigeminal neuralgia, and SUNCT syndrome (*s*hort-lasting, *u*nilateral, *n*euralgiform headache with *c*onjunctival injection and *t*earing). Various treatment modalities have been used with varying degrees of success. These include glycerol injection, local anesthetic blockade, trigeminal ganglion ablation or decompression, acupuncture, and neurolysis.[2] In rare instances, a discrete neuroma can be identified and resected to good therapeutic end.[3–5] In other cases, the supraorbital and/or supratrochlear nerves bear no distinct lesion but may be entrapped in scar tissue or suffer from compression by another source that leads to a chronic and severe headache syndrome.[6] These patients have often undergone extensive diagnostic testing or have taken numerous neuroleptics or pain relievers without benefit.

Surgical management of this syndrome has been previously reported in the literature; how-

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ever, the details of the operation have been vague[6] or the patient series has been limited.[2] We present a series of six patients diagnosed by a neurologist as suffering from posttraumatic supraorbital neuralgia. These patients underwent surgical excision of the supraorbital and supratrochlear nerves. As a technical refinement of the procedure, the cut, adjacent proximal stumps of the supraorbital and supratrochlear nerves were directed toward each other in a loop fashion by means of a neural tube conduit. Outcomes are discussed.

PATIENTS AND METHODS

A retrospective chart review was conducted for the period between February of 2005 and April of 2007. A series of six consecutive cases of patients presenting to the senior author (I.D.) and undergoing supraorbital and supratrochlear nerve excision and implantation for posttraumatic headache attributed to supraorbital neuralgia are reported. All patients underwent an identical procedure. Institutional review board approval was obtained. No patients meeting the above criteria were excluded.

Pain was assessed using a visual analogue pain scale ranging from 0 to 10, with 0 being no pain and 10 being the worst pain imaginable. Preoperative and postoperative pain levels were compared. A Wilcoxon signed rank test was used for statistical analysis. Therapeutic benefit of the procedure was defined as a reduction of pain by 50 percent. Mean follow-up was 14 months. Patients were also asked to subjectively rate their quality-of-life improvement as a result of surgery. A summary of the cases is presented in Table 1.

In each case, the patient had undergone numerous failed treatment modalities. These included injection with local anesthetic and steroids, and pharmacotherapy. All patients had tried either tricyclic antidepressants, carbamazepine, or ergot derivatives to minimal therapeutic effect. All patients were dependent on narcotic pain medication. In addition, several had had multiple diagnostic tests, including brain computed tomography and magnetic resonance imaging, the results of which had been negative.

Patients consisted of five women and one man. The mean age of the patients was 42 years. All patients could identify an inciting traumatic or surgical event immediately before or several months before the onset of headaches. All were unilateral. The pain distribution was reported to be over the forehead with radiation into the scalp and upper eyelid. The pain was rated as fluctuating from moderate to severe, often causing difficulties in work or social performance. The pain was chronic in nature, with attacks lasting days to weeks with occasional brief respite. It was often described as being dull and constant without occasional "stabs." Two patients experienced photophobia. No patients experienced aura, conjunctival injection, or rhinorrhea. No trigger mechanisms were identified.

On examination, all patients were tender to palpation over the exit of the supraorbital and supratrochlear nerves at the supraorbital rim on the affected side, although a distinct Tinel's sign was not tested. Firm pressure in this area could duplicate the headache pain. The unaffected side was nontender. Sensation in the distribution of these nerves was grossly normal to mildly diminished in three patients, and two had paresthesias or numbness. Sensitive monofilament or two-point discrimination testing was not used. All patients had undergone preoperative supraorbital and/or supratrochlear nerve blockade with effective but short-lasting pain alleviation.

Operative technique consisted of, under general anesthesia, dissection of the supraorbital and/or supratrochlear nerves on the affected side by means of an upper lid incision after careful local infusion of 1% lidocaine with epinephrine. Sharp incision is made through the skin and bipolar cautery is used to carry the dissection in anatomical layers through the orbicularis muscle

Table 1. Patient Population

Age (yr)	Sex	Mechanism	Side	Preoperative Pain*	Postoperative Pain*	Improvement (%)	Follow-Up (mo)	QOL Improvement (%)
37	F	Tumor removal	R	10	0	100	26	100
46	F	Forehead laceration	R	10	2	80	22	90
46	F	Blunt assault	L	10	0	100	13	100
46	F	Brow lift	R	8	2	75	9	90
50	F	MVC	L	7	5	28	7	0
27	M	Blunt assault	R	10	0	100	8	100

F, female; M, male; R, right; L, left; MVC, motor vehicle collision; QOL, quality of life (subjectively rated by patient at postoperative visit).
*Pain measurements are based on a visual analogue scale ranging from 0 to 10, with 10 being the worst pain imaginable.

onto the orbital septum. Spreading with tenotomy scissors superiorly toward the orbital rim then identifies the corrugator muscle and nerves. The nerves, dissected under loupe magnification, were often found to be encased in scar tissue. No discrete neuromata were identified intraoperatively, although distal dissection to areas involved in scar was not used. At a point 2 to 3 cm distal to the exit from the frontal bone, a 1- to 2-mm segment of the supraorbital and supratrochlear nerves was sharply resected. To minimize the risk of future neuroma formation, in a novel approach, the cut adjacent proximal nerve stumps were then directed toward each other in a loop fashion and microsurgically implanted end to end in a neural tube conduit. Meticulous hemostasis followed by layered closure was then undertaken (Fig. 1).

RESULTS

Of a total of six patients, five (83 percent) experienced significant, durable pain relief of at least 50 percent or greater. Three of these patients were completely pain free following this procedure. There was one treatment failure, with only a modest improvement in the visual analogue pain score and without quality-of-life improvement. Preoperative average pain was 9.16 ± 1.3 and postoperative average pain was 1.5 ± 1.9, a reduction of 7.7 points (83 percent) ($p = 0.03$). All patients were appropriately insensate within the distribution of the supraorbital and supratrochlear nerves. No operative complications were encountered. Pathology reports were available for three of the six patients. These indicated either perineural fibrosis or microscopic neuroma.

DISCUSSION

The trigeminal nerve arises from the lateral side of the pons. The sensory root gives rise to the gasserian ganglion, which lies within the petrous part of the cranial bone in the posterior cranial fossa. The first division of the trigeminal nerve, the ophthalmic nerve, arises from the anteromedial gasserian ganglion and then passes through the wall of the cavernous sinus below the trochlear nerve.[7] It then divides into the frontal, lacrimal, and nasociliary branches, which enter the orbit by means of the superior orbital fissure. The frontal branch lies just above the levator complex and divides into the supraorbital and supratrochlear nerves, typically before exit from the skull. These provide sensation to the skin of the forehead, the medial angle of the eye, and upper eyelid but not the cornea.[2] The supraorbital nerve typically exits the frontal bone by means of a palpable notch or foramen along the medial third of the supraorbital rim; however, this exit may vary in rare instances, to be located anywhere along or above or even lateral to the supraorbital rim.[8] The supratrochlear nerve typically leaves no notch or

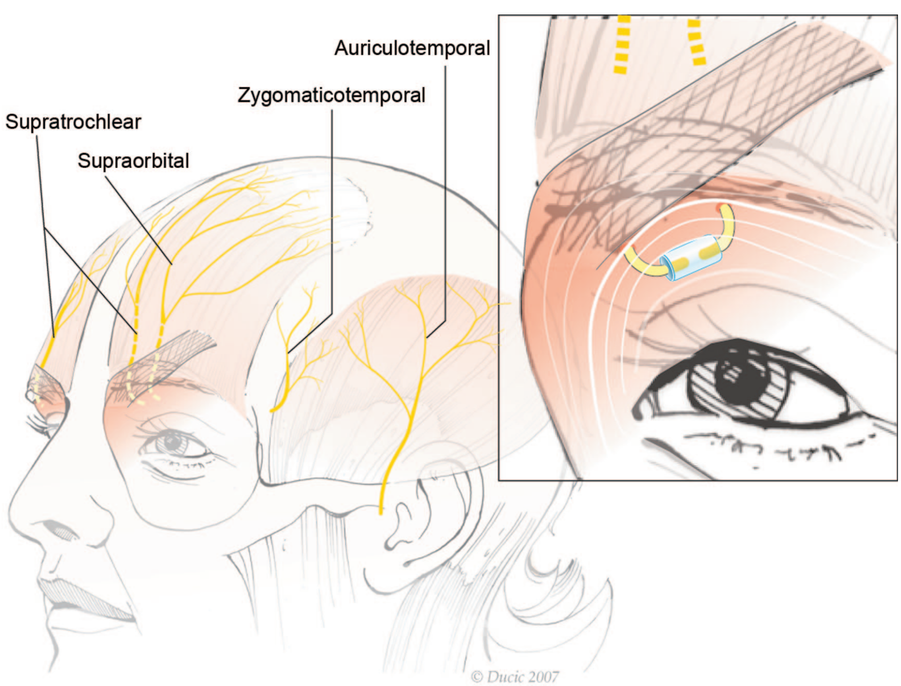

Fig. 1. Anatomy of the supraorbital and supratrochlear nerves. *Inset* demonstrates coaptation of the proximal nerve ends by means of a neural tube conduit.

impression on the frontal bone. Beyond the orbital rim, the supraorbital nerve has two consistent divisions; a superficial or medial division that passes above the frontalis muscle to innervate forehead skin and the anterior margin of the scalp, and a deep or lateral division that travels distally along the lateral forehead between the galea and the pericranium.[9] Clinically, the supraorbital nerve can be located by means of palpation with the fingernail or along a line in the midsagittal plane perpendicular to the edge of the medial limbus. The supratrochlear nerve is located between 8 and 10 mm medial to the supraorbital nerve.[10] It is this superficial location, with minimal soft-tissue coverage and abutting the noncompressible frontal bone, that renders the supraorbital and supratrochlear nerves susceptible to injury.

Supraorbital neuralgia is a distinct clinical entity, although it shares similar features with a number of other headache syndromes: cluster headache, migraine, SUNCT syndrome, and trigeminal neuralgia. For this reason, the nomenclature and diagnosis can be confusing. Although the pain of supraorbital neuralgia is often unilateral and periorbital as it is in cluster headache, supraorbital neuralgia lacks the autonomic features of ptosis, conjunctival injection, lacrimation, facial flushing, and rhinorrhea, although photophobia may be present.[11] Isolated V_1 trigeminal neuralgia, or tic de Loreux, is felt to be exceedingly rare, constituting only 4 percent of trigeminal neuralgia cases in one study.[12] Trigeminal neuralgia episodes are typically unilateral and brief (<1 minute), with variable frequency. Autonomic symptoms are occasionally present, and distinct trigger mechanisms are frequently identified.[13] Disease is attributed to a lesion of the trigeminal ganglion. By contrast, supraorbital neuralgia is characterized by episodes of longer duration (hours to months) and the abnormality is felt to lie with the peripheral nerve itself. The term SUNCT represents short-lasting, unilateral, neuralgiform headache with conjunctival injection and tearing.[14] In this syndrome, the pain is generally less intense than in trigeminal neuralgia, and unlike trigeminal neuralgia, SUNCT does not respond to supraorbital nerve blockade.[13] Again, supraorbital neuralgia is characterized by longer attacks without autonomic symptoms. Finally, migraine headaches may have a prodromal aura and throbbing character and tend to be more episodic and self-limited in nature, with variable unilaterality or bilaterality.[15] To further confuse the issue, however, supraorbital nerve entrapment may be implicated in triggering migraine type pain.[16] Furthermore, supraorbital neuralgia may be confused with chronic frontal sinusitis.[17]

Diagnostic criteria for supraorbital neuralgia have been elucidated previously.[18] These include the triad of unilateral headache without side shift, fronto-ocular area pain, and local tenderness at the supraorbital notch or foramen. Other implicating factors include lack of infraorbital nerve tenderness, forehead tenderness on the symptomatic side, and mild sensory disturbance on the ipsilateral forehead.

The prevalence of supraorbital neuralgia has been estimated to be 0.5 percent in one epidemiologic study.[18] In this study, 83 percent of those with supraorbital neuralgia could report an inciting forehead trauma. The pain was typically mild to moderate; however, this reflects the fact that these individuals were discovered in a population survey and were not seeking treatment for their pain. The prevalence of severe supraorbital neuralgia requiring medical treatment is not known.

Oftentimes, patients with supraorbital neuralgia have undergone other pharmacologic or more invasive treatments to alleviate their symptomatology. This was the case in our cohort. This fact illustrates the need for a broader understanding of the differential diagnosis of frontal headache, as the treatments for each entity vary greatly. Glycerol injection, ganglionic section, thermocoagulation, and the Janetta procedure may be of therapeutic benefit in trigeminal neuralgia but are often too radical or invasive to be appropriately considered in supraorbital neuralgia.[2] Similarly, pharmacologic management appropriate for migraines, cluster headaches, and SUNCT syndrome is of scant benefit in supraorbital neuralgia.[6]

Multiple reports exist in the literature of posttraumatic supraorbital neuralgia. Occasionally, a neuroma is found at the time of exploration or is clinically palpated preoperatively. Reported events leading to neuroma of the supraorbital or supratrochlear nerves include brow lift,[3] motor vehicle collision,[19] shrapnel injury,[4] orbital enucleation,[20] halo fixation,[5] frontal bone fracture,[21] swim goggle use,[22] and even insect bite.[23] In these cases, resection of the neuroma has been curative. Purely compressive causes including "swim goggle headache,"[24,25] postoperative scarring,[19,26] anesthesia mask,[27] cryptic hemangioma,[28] blunt trauma,[18] and idiopathic causes[29] have also been described.

Reports of successful treatment of supraorbital neuralgia in the absence of palpable neuromas are scarce. Acupuncture has been reported to be of some therapeutic benefit.[30] Local anesthetic injection of the supraorbital nerve alone has been reported to be curative[26,29]; however, these injections often provide relief of short duration and must be repeated frequently.[6] Supraorbital nerve avulsion has been used; however, symptoms tend to recur in 6 to 12 months.[2] There is a report of a series of five patients who underwent surgery for supraorbital neuralgia of unknown ($n = 4$) or posttraumatic ($n = 1$) causes.[6] The surgical details are vague, referencing either nerve exploration or division of "a constricting band." In this series, two of five patients suffered recurrence of their pain postoperatively after a symptom-free interval. One patient underwent reexploration and was found to have scar tissue, which was removed, affecting a more lasting cure. An intraorbital approach to division of the supraorbital nerve has been described to overcome the possibility of recurrence resulting from postoperative scaring in a series of four patients, with pain-free success in three[7]; however, this procedure can endanger the orbital contents and results in variable postoperative ptosis. One case report exists of successful treatment of supraorbital neuralgia following blunt trauma by means of an endoscopic approach.[19]

In our series, a novel approach to the management of posttraumatic supraorbital neuralgia has been effective. The success of this procedure, and that of other surgical approaches,[6,7,19] implicates compressive abnormality within the surrounding soft tissues of the supraorbital region or abnormality within the nerve itself. In our series, the assumption was made at the outset that, although no neuromata were palpable, these nerves were likely to be affected by internal abnormality, neuroma-in-continuity given their traumatic onset. We therefore treated these lesions as neuromas and excised the nerves as opposed to performing external neurolysis alone. Because little muscle bulk is available in this region for nerve implantation following resection, the end-to-end coaptation of the cut nerve ends with a neural tube was used to minimize the risk of recurrence caused by neuroma or postoperative scarring. In particular, this is important in patients who have a traumatic cause of their pain, as the local tissues tend to be damaged and prove an inhospitable healing environment were excision or neurolysis alone to be used. In contrast to more centrally directed procedures, corneal sensation is preserved and risk of damage to the orbital contents is minimized.

It is unclear why the one treatment failure occurred. This patient was pain free for several weeks after the procedure; however, her pain ultimately returned only modestly diminished. We postulate that the high-energy nature (motor vehicle collision) of the inciting injury could have caused additional lesion of the nerve more proximally, perhaps within the orbit itself. Similarly, in this case, extensive scarring was noted along the supraorbital rim. It is also possible that this scarring reformed to compress the remaining nerve stump. Perhaps a neuroma reoccurred as well, following the degradation of the neural tube. We feel that this patient is not malingering to receive narcotics, as she is prescribed narcotic pain medication for chronic pain elsewhere. This patient is currently considering surgical reexploration.

With regard to a management strategy, we suggest evaluation by a neurologist preoperatively. Magnetic resonance imaging may be used if a mass lesion is suspected. Meticulous inspection for cutaneous malignancy must be performed.[31] Conservative management should be attempted for at least 6 months before the procedure with either pain medication or local injection, as this problem may be self-limited. Failing conservative management, the patient must be counseled that they will be permanently insensate along the forehead and upper eyelid on the affected side. Often, they find this condition preferable to chronic pain.

Clearly, this report suffers from limited sample size and relatively short follow-up duration. Without a control, it is impossible to negate the possibility of placebo effect; however, sham surgery here is out of the question. It remains to be seen how durable the pain relief is. The dramatic "life-changing" improvement in these patients seems quite promising. In the future, we hope to present a more extensive cohort. It is not known whether neurolysis alone would have proven equally effective. This seems unlikely, however, given the extensive scarring encountered, the consequent internal neural lesion that this implies, and the tendency of pain recurrence attributable to this. Similarly, in the setting of failed neurolysis, difficult identification of the nerve, not to mention the cost of a repeat procedure, make neurolysis alone less desirable. From this limited experience, however, it appears that this procedure is safe and effective.

Posttraumatic headache in the distribution of the supraorbital or supratrochlear nerve can be a debilitating problem. Few effective treatment strategies have been described. This experience with nerve excision and end-to-end coaptation of the proximal nerve stumps appears to present an effective modality with which to alleviate the headache symptoms in these patients.

CODING PERSPECTIVE

This information prepared by Dr. Raymond V. Janevicius is intended to provide coding guidance.

64910 Nerve repair; with synthetic conduit or vein allograft (e.g., nerve tube), each nerve

- Code 64910 is a relatively new code which first appeared in CPT in 2007. It includes preparation of nerve ends prior to use of the conduit.
- Although two different nerves are coapted with the conduit, code 64910 is only reported once, even though the descriptor indicates "each nerve." Only two nerve ends are coapted, making the procedure described comparable to the bridging of a gap in a single nerve.
- Unlike other neurorrhaphy codes (64831 to 64876), code 64910 *includes* use of the operating microscope. Code 69990 is not reported in addition to 64910.

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