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Expert Opinion

Supraorbital Neuralgia

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Supraorbital neuralgia is defined by the International Classification of Headache Disorders-2 by the following diagnostic criteria: paroxysmal or constant pain in the region of the supraorbital notch and medial aspect of the forehead in the area supplied by the supraorbital nerve; tenderness over the nerve in the supraorbital notch; and pain is abolished by local anesthetic blockade or ablation of the supraorbital nerve.[1]

CLINICAL HISTORY

A 45-year-old male presented following a workplace accident 3 months prior. He had been struck by a 2-pound metal hook across the right forehead with loss of consciousness for about 30 seconds. A small laceration of the right eyebrow had been sutured in the emergency room. Following the occurrence of posttraumatic headaches, the patient then saw a primary care physician who prescribed naproxen sodium, and then a butalbital, acetaminophen, caffeine, and codeine combination tablet without benefit. A CT scan of the brain and orbits was normal.

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He reported a constant aching and throbbing pain over the right eyebrow with an intensity ranging from 3-8/10 with an average of 5-6/10 with throbbing at times over the right forehead. The patient also experienced occasional nausea, difficulty with vision focus, and some short-term memory problems but no dizziness, hearing loss, tinnitus, or loss of smell or taste.

There was a 15-year history of chronic migraine without aura, which had much improved when started on venlafaxine and nadolol for prevention with only 2 in the prior 3 months. There was no prior history of significant head injury. He had not been able to return to work.

On examination, there was a scar over the right eyebrow and a positive Tinel's sign over the right supraorbital nerve at the supraorbital notch. Neurological examination was normal except for decreased sensation in the distribution of the right supraorbital nerve.

The patient was placed on pregabalin 75 mg twice daily for 1 week and then 150 mg twice daily. When seen for follow-up 3 weeks later, the headache was only intermittent and was brought on by movement of the head or focusing of the eyes with an intensity of 5-7/10 lasting for perhaps 2 hours. He reported no side effects of pregabalin. His memory had returned to normal. Examination revealed the same positive Tinel's sign and decreased sensation in the right supraorbital nerve distribution. The dose of pregabalin was increased to 300 mg twice daily.

Questions.—What are the causes and symptoms of supraorbital neuralgia? What medications are effective? Are nerve blocks effective? What is the prognosis?

EXPERT OPINION

There is little doubt the patient is suffering from supraorbital neuralgia. The combination of pain in the territory supplied by the supraorbital nerve, Tinel's sign at the supraorbital notch, and hypoesthesia in the forehead, are all features pointing to a neuropathic pain stemming from the supraorbital nerve.[1,2] Moreover, according to the reported traumatic event the nerve was presumably injured at its emergence in the forehead.

The forehead is innervated by the supratrochlear and supraorbital nerves, both stemming from the frontal nerve, one of the terminal branches of the first division of the trigeminal nerve. The frontal nerve runs forward in the superior wall of the orbit and divides into supratrochlear and supraorbital nerves that exit the superior edge of the orbit, frequently passing through an incisure or foramen (notch), and supply the medial and lateral aspects of the forehead respectively.

What are the Causes and Symptoms of Supraorbital Neuralgia?—In 2 hospital-based studies of supraorbital neuralgia the disorder was generally primary.[2,3] In a large epidemiological study, however,[4] most patients had a preceding forehead trauma. Symptomatic supraorbital neuralgia cases are usually posttraumatic,[4,5] and less frequently secondary to tumors[6,7] and infections.[8]

In the forehead, the nerve runs superficially and may be easily damaged by a major external force against a hard surface (frontal bone)[4,5] or during surgery.[9] With major trauma antecedents, the patients have good recall of the event and report that the symptoms usually started soon after the injury, whereas minor trauma could be either unnoticed or underreported. When the patient can hardly relate the actual symptoms to a possible past trauma, finding a scar and a sensibility loss in the forehead retrospectively suggest a traumatic etiology.[10] Otherwise, the differences in the delay of posttraumatic symptoms may also indicate different types of nerve injury: acute trauma with direct injury resulting in neurapraxia and neurotmesis would produce early onset of symptoms, whereas subtle/persistent trauma, fibrosis and neuroma formation would result in a delayed onset of symptoms with rather chronic course. Anyway, recovery would depend on the degree of nerve damage.

Application of external, brief, pressure in the head, such as those produced by a band around the head, a tight hat, oxygen and anesthetic masks, or swimming goggles, may give rise to a headache that resolves once the external pressure has been removed.[11] The headache may result from a continued stimulation of cutaneous nerves of the head in patients with a premorbid suceptibility to nerve compression. It is an in crescendo, non-pulsating headache without accompaniments, with maximum pain in the points of more external compression. Involved nerves in such a type of headache seem to be mostly the supraorbital and supratrochlear ones.

Some "primary" supraorbital neuralgia may be due to subtle microtrauma. Progressive slow lesion can be produced at sites where the nerve is confined to narrow anatomical passageways such as the supraorbital notch and inside the orbit where the nerve may be indirectly influenced by eyeball movements. In the supraorbital notch a chronic compression by bony excrescencies or tissue bands may occur.[2] Moreover, the supraorbital artery accompanies the supraorbital nerve passing through the supraorbital notch, where even the nerve and the vessel may be interwoven, so, an impingement of the nerve by vascular engorgement could theoretically occur.[2,3]

In the orbit, the supraorbital nerve – before exiting the orbital edge, shows an intimate relation with nearby vessels, oblique superior muscle, lacrimal gland, and the eyeball itself. We have postulated that alongside such intraorbital traject the supraorbital nerve may be prone to intermittent subtle trauma, during ocular movements, that would produce either stretching, angulation, traction, or friction of the nerve trunk. Since the nerve and a small artery can be very close, an adjacent artery might produce a microvascular compression of the nerve. Also, movements of the superior oblique muscle might pull the nerve itself, either directly or indirectly through adja-

cent stuck tissues. Otherwise, the nerve may also be involved in structural or inflammatory processes of the orbit.

Supraorbital neuralgia is a rare disorder of adulthood. Primary cases are slightly predominant in the female,[2,3] whereas the posttraumatic variety seems to prevail in the male.[4] The age at onset is around 30-40 years.[2-6] Clinically it is characterized by a triad: (1) forehead pain in the territory supplied by the supraorbital nerve without side shift; (2) tenderness on either the supraorbital nocht or traject of the nerve; (3) absolute – but transitory – relief of symptoms upon supraorbital nerve blockade.[2] Such 3 cardinal features have been selected as diagnostic criteria by the IHS.[1] The pain may be severe – typically continuous, with paroxysmal exacerbations, and usually lacking autonomic accompaniments. Interestingly, the pain may be precipitated or exacerbated by frowning. In addition, there may be signs and symptoms of sensory dysfunction (hypoesthesia, paresthesia, and allodynia), and typical "neuralgic features" (lightning pain and local, exteroceptive precipitating mechanisms). However, both sensitive and neuralgic features are not constantly present and seem to be more frequent in the secondary – usually posttraumatic – forms.[5] Another important issue is that posttraumatic supraorbital neuralgia may be part of the posttraumatic syndrome, which includes symptoms such as concentration difficulties, memory problems, emotional lability, irritability, insomnia, fatigue, disequilibration, dizziness, etc, presenting in various combinations.

What Medications Are Effective?—Pharmacological treatment is based on antineuralgic drugs. In particular, gabapentin (800-2400 mg daily) or pregabalin (150-300 mg daily) and amitriptyline (25-75 mg daily) proved to be of avail in some patients.[2-5] Capsaicin topically applied on the symptomatic forehead has also rendered substantial benefits in some patients.[12] Therapy may include surgical exploration of the supraorbital notch and eventual liberation of the nerve from tissue bands or bone excrescencies. This procedure has been remarkably successful in the group of patients reported by Sjaastad et al.[2] Invasive procedures such as nerve rhizolisis, radiofrequency lesioning, cryodenervation, even surgical ablation of the nerve may be necessary to abolish refractory, unbearable, pain. Peripheral nerve stimulation of the supraorbital nerve has recently been reported with promising results.[13]

Are Nerve Blocks Effective?—Anesthetic blockade of the supraorbital nerve provides temporary relief. It is more a diagnostic tool than a therapeutic resource. Admittedly, repeated anesthetic blocks or a single shot with a mixture of local anesthetic and long-acting steroid may provide a rather sustained relief with obvious therapeutic implications.

What is the Prognosis?—Posttraumatic supraorbital neuralgia seems to have a rather good prognosis since in most reported cases the symptoms disappeared within one[6] – some[5] years, although in several ones hypoesthesia persisted. Primary cases tend to display a prolonged, chronic evolution.

Follow-up.—When the headache was no better 3 weeks later, he received a right supraorbital nerve block with 1 cc of 1% lidocaine with great relief for about 2 weeks but then recurrence. Three weeks later, he received a second block with 0.5 cc of lidocaine and 0.5 mg of triamcinolone with improvement for 1 week. He then received 3 more blocks with 1 cc of 1% lidocaine alone spaced 3-4 weeks apart. Seven months after the last injection (about 15 months after the injury), the pain was still fairly constant but much less with a level of 2-3/10 and about once a week 6/10 on pregabalin 300 mg at bedtime (the patient had decreased the dose from twice a day to once a day on his own). The pain intensified while bending over or looking up. On examination, there was still numbness in the right supraorbital nerve distribution.

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