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CLINICAL CORRESPONDENCE

Idiopathic trigeminal neuropathy may respond to greater occipital nerve injection

MW Weatherall

Princess Margaret Migraine Clinic, Imperial Healthcare NHS Trust, Charing Cross Hospital, London, UK

Mark W. Weatherall, Princess Margaret Migraine Clinic, Imperial Healthcare NHS Trust, Charing Cross Hospital, Fulham Palace Road, London W6 8RF, UK. E-mail mark.weatherall@doctors.org.uk *Received 29 July 2007, accepted 15 December 2007*

Trigeminal neuropathy is a condition characterized by sensory disturbance in the distribution of the trigeminal nerve (1). It can be caused by a wide variety of conditions, including trauma, tumours, connective tissue disorders, infections or neurovascular conflict. In many cases, however, no obvious cause is found. A case is presented in which a patient with idiopathic trigeminal neuropathy underwent ipsilateral greater occipital nerve injection with significant improvement in symptoms.

Case report

A 69-year-old woman presented in May 2006 with a 2-year history of intermittent sensory disturbance of the left side of her face. Initially these symptoms were intermittent and infrequent, but by the time of review the attacks were happening on an almost daily basis, lasting between 30 min and 24 h. She described a sensation like a local anaesthetic over the left cheek, initially in the area below the eye with occasional numbness and tingling affecting her left upper lip. This sensation might build up in intensity over some hours, although was never terribly uncomfortable. She was also aware of some discomfort in the left occipital region, radiating to the left side of her neck.

On direct questioning she admitted to some dryness of the mouth, but no dryness of the eyes or constipation. Her hearing was normal. There was no history of diplopia or visual blurring. She had a past history of infrequent migraine without aura, which had settled in her 40s. On occasions her blood pressure had been found to be slightly elevated, but she was on no medication for this. She was a non-smoker and non-drinker. Her weight and appetite had been normal.

On examination her fundi and cranial nerves were normal. The corneal reflexes were present, equal and normal, and there was no evidence of impairment of sensation in any of the branches of the trigeminal nerve on the left. However, there was some tenderness in the upper cervical region on the left, and some pain on neck movement. The rest of the neurological examination was normal.

Initial investigations revealed a normal full blood count, serum biochemistry and serum protein electrophoresis. Antinuclear antibodies and extractable nuclear antigens were negative. The erythrocyte sedimentation rate was 12 mm/h. Magnetic resonance imaging (MRI) of the brain showed a few high signal white matter lesions, consistent with small vessel ischaemia. MRI of the cervical spine showed degenerative changes at C4/5, C5/6 and C6/7, but no evidence of root compression or any cord abnormality. A diagnosis of idiopathic trigeminal neuropathy was made.

When reviewed in January 2007, she continued to complain of sensory disturbances, now affecting the whole of the distribution of V2 on the left, extending to the nose and to the eye on that side, and more severe in that she found it difficult to ignore them and continue her normal daily activities. Once again there was no objective evidence of sensory loss in this distribution. There was, however, definite tenderness of the greater occipital nerve on that side. The patient was reluctant to take medication, but agreed to a greater occipital nerve injection.

With her consent, the left greater occipital nerve was infiltrated with 1 ml 1% lidocaine and 40 mg Depo-medrone. There were no immediate side-effects.

She was reviewed 8 weeks later. She had experienced a very positive response to the injection. Her occipital discomfort had settled. Her facial sensory disturbances were much less severe, and could now be ignored if necessary. They were also much less extensive, now being confined simply to

the original affected area in her left cheek. Examination revealed mild tenderness of the left greater occipital nerve, and a second injection of lidocaine and Depo-Medrone was performed. When reviewed 3 months later, there had been no further improvement, although her symptoms had remained largely unchanged.

Discussion

This is the first report of successful treatment of the symptoms of idiopathic trigeminal neuropathy with greater occipital nerve injections. The first report of the use of greater occipital nerve injection in the management of headache disorders appeared over 60 years ago (2). In recent years, the effectiveness of this intervention has been confirmed in the treatment of several primary and secondary headache disorders, including migraine (3, 4), cluster headache (5–8), hemicrania continua (4), occipital neuralgia (3) and cervicogenic headache (6). The pathophysiological basis for its effectiveness is not clear, but is believed to relate to the modulation of input into second order neuronal processing in the trigeminocervical complex.

The trigeminocervical complex comprises the trigeminal nucleus caudalis in the caudal medulla and the neurons of the dorsal horns at C1 and C2 (9–11). Experimental stimulation of structures innervated by the trigeminal nerve, such as the superior sagittal sinus and middle meningeal artery, can activate neurons in this complex (9, 12). Efferents from the complex synapse in the superior salivatory nucleus; experimental stimulation of the superior sagittal sinus activates neurons in this nucleus, suggesting that this pathway is relevant in the production of pain in headache disorders (13). Parasympathetic fibres run from the superior salivatory nucleus to the sphenopalatine ganglion, mediating the parasympathetic features of primary headache disorders and vasodilation of both cerebral and extracerebral arteries, thus completing the trigeminovascular feedback loop that exacerbates vasodilation and pain in these disorders (14, 15). The fibres of the greater occipital nerve, which is the main sensory nerve of the posterior region of the head, originate predominantly from the C2 dorsal root (16). Stimulation of the greater occipital nerve also activates neurons in the trigeminocervical complex, and in some cases also causes ipsilateral conjunctival injection, eye watering, and ptosis (17, 18). It is clear, therefore, that an anatomical pathway links C2 activation to the trigeminocervical complex, and thereby to the superior salivatory nucleus, sphenopalatine ganglion, and the other structures involved in mediating headache disorders.

Although the symptoms of idiopathic trigeminal neuropathy may undergo spontaneous remission, they often persist for many years (19). Symptoms respond to medications commonly used for neuropathic discomfort, such as tricyclic antidepressants, gabapentin or pregabalin, but many patients find the side-effects of such medications limit their effectiveness. Greater occipital injection is simple and safe, the only significant reported side-effect being localized alopecia and cutaneous atrophy (20), and would be a useful addition to the treatment possibilities for this condition. The presence of greater occipital nerve tenderness in patients with trigeminal neuropathy has not been noted before. This is perhaps because it has not been looked for. A systematic evaluation of the presence of this symptom in trigeminal neuropathy would be useful, as would a controlled trial of the effectiveness of greater occipital nerve injection in patients with trigeminal neuropathy, both with and without ipsilateral greater occipital nerve tenderness.

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