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## BRIEF REPORT

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# Great occipital nerve blockade for cluster headache in the emergency department: case report

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L. Scattoni (⊠) • F. Di Stani • V. Villani D. Dugoni • C. Mostardini • C. Reale R. Cerbo Pain Center "Enzo Borzomati" Policlinico Umberto I University of Rome "La Sapienza" v.le del Policlinico 155, 00185 Roma e-mail: lorescat@yahoo.it Tel.: 06-49979190 Fax: 06-49979191 Abstract A 44-year-old man with a past medical history of episodic cluster headache presented in our ED with complaints of multiple daily cluster headache attacks, with cervico-occipital spreading of pain from May to September 2004. The neurological examination showed no abnormalities as well as brain and spine MRI. Great Occipital Nerve (GON) blockade, with Lidocaine 2% (5 ml) and betamethasone (2 mg), were performed in the right occipital region (ipsilaterally to cluster headache), during attack. GON blockade was effective immediately for the attack and the cluster period resolved

after the injection. We suppose that the action of GON blockade may involve the trigemino-cervical complex and we moreover strongly suggest to use GON blockade in emergency departments for cluster headache with cervico-occipital spreading as attack abortive therapy, especially in oxygen and sumatriptan resistant cluster headache attacks, in patients who complaints sumatriptan side-effects or have contraindications to use triptans.

**Key words** Great occipital nerve blockade • Cluster headache • Emergency department

### Introduction

Cluster headache (CH) is characterised by attacks of severe unilateral pain in the orbital, supraorbital and/or temporal areas, lasting from 15 up to 180 min, recurring up to 8 times daily and accompanied by ipsilateral autonomic symptoms. In the episodic form, headache attacks usually occur in bouts (cluster periods) lasting from one week to 1 year, separated by pain-free periods of at least 1 month. In the chronic form these pain-free periods are absent or last less than a month [1]. Although effective acute treatments are available for CH attacks, e.g., subcutaneous sumatriptan injections, most patients need, in addition, preventative therapy. Several drugs, such as verapamil, methysergide and lithium carbonate, have proven efficacious in the prevention of CH attacks and shortening of bouts. Oral steroids are probably the most effective short-term preventative treatment [2], but patients may become steroid-dependent and develop serious steroid-related adverse effects within a few months.

Anthony [3] shows that suboccipital injections of a local anaesthetic alone have neither a beneficial nor a

worsening effect on CH attacks and 0.5 ml xylocaine 2% was also added to the steroid preparation. Ambrosini et al. [4], in a placebo-controlled study, showed that suboccipital injection with a mixture of rapid- and long-acting steroids and xylocaine 2%, which can be used as a single-shot treatment, is a more effective modality for steroid therapy of episodic and chronic CH.

We report the case of a 44-year-old man with a history of episodic CH treated in the emergency department (ED) with an injection of a mixture of local anaesthetic and steroid, with complete pain relief lasting one year. We suggest the use of this therapy on patients with oxygenand sumatriptan-resistant CH attacks in the ED and in patients who complain of sumatriptan side effects or have contraindications to triptans [5, 6].

#### **Case report**

A 44-year-old man with a past medical history of episodic CH presented in our ED with complaints of multiple daily CH attacks lasting from May to September.

The pain was described as burning, strictly confined to the right retro-orbital region, spreading to the right occipital region and usually lasting from 45 min to 3 h. Mean headache intensity was 10 (Numerical Rating Scale; 0 = no pain, 10 = the worst pain imaginable). The headaches were associated with ipsilateral rhinorrhoea, eyelid ptosis, conjunctival congestion, lacrimation, restlessness and a sense of agitation.

Because the headache became daily with multiple attacks and the pain worsened, he frequently went to the ED (8–10 times) between May and September 2004. Previous therapy based on methylprednisolone (16 mg bid) and valproic acid (500 mg/day) was administered by another headache centre without any success; O<sub>2</sub> therapy, indomethacin (50 mg) and rizatriptan were ineffective for the patient's attacks, and only subcutaneous sumatriptan was effective, until the end of May 2004 when it became uneffective.

In September 2004 he visited our ED and was examined by a neurologist. The neurological examination showed no abnormalities, as did brain and spine MRI and ECG. After informed consent, we performed great occipital nerve (GON) blockade, with lidocaine 2% (5 ml) and betamethasone (2 mg), in the right occipital region (ipsilaterally to CH), during an attack; the injection of supraorbital and maxillary branch of trigeminal nerve was impossible because the patient was restless because of the pain. GON blockade was defined as the appearance of hypo-anaesthesia in the GON area after the procedure. Clinical response was good: he was pain free in a few minutes and preventive therapy, started in the previous Headache Center in August 2004, was not modified. The patient did not show any side effects after the injection. At the one-month follow-up visit he reported strong headache improvement in frequency and intensity from 10 to 1–2 on the NRS and then we gradually stopped preventive therapy. At the one-year follow-up visit (September 2005) he was still pain free.

#### Discussion

As described by Ambrosini et al. [4], we observed that the GON blockade was effective by itself in stopping cluster symptomatology, while our patient was experiencing multiple cluster attacks per day. We do not know if the cluster period was spontaneously ending at the time of injection, but it is improbable because the frequency of the attacks was increasing. Moreover the temporal relationship between GON blockade and cluster symptomatology cessation was very strong.

The mechanisms of GON blockade efficacy in CH are unknown. Busch [7] showed a functional connectivity between cervical pathway and trigeminal sensory afferents in healthy subjects in a neurophysiological study. He also demonstrated that it is possible to modulate the trigeminal nociceptive system by GON blockade.

As a result, we suppose that the action of GON blockade in CH with cervico-occipital spreading may involve the trigemino-cervical complex [8], an overlapping area between trigeminal and cervical sensory afferent projections in the central nervous system, producing central desensitisation of this area and, probably, altering the trigeminal autonomic reflex pathway [9]. This is probably the mechanism that would justify the use of GON blockade as a transitional preventive therapy and it may explain the efficacy in our patient as a single-shot therapy.

Rozen [10] suggested that there can be a non-hypothalamic form of CH and that the GON may play a significant role in cluster pathogenesis in some patients. The trigeminocervical-hypothalamic connection could secondarily activate the hypothalamus.

There is actually no current gold standard of practice regarding GON injections in the management of primary headache (PH). Recently, Afridi et al. [11] showed that tenderness over the GON is strongly predictive of outcome after GON blockade in PH as CH.

In conclusion we strongly suggest the use of GON blockade in EDs for CH with cervico-occipital spreading as attack abortive therapy, especially in oxygen- and sumatriptan-resistant CH attacks, in patients who complain of sumatriptan side effects or have contraindications to use of triptans (ischaemic heart disease, obliterating arteriopathy of the lower extremities, prior acute cerebrovascular disease, severe liver failure or uncontrolled arterial hypertension) [5].

## References

- 1. Headache Classification Subcommittee of the International Headache Society (2004) The international classification of headache disorders, 2nd edition. Cephalalgia 24[Suppl 1]:1–160
- Ekbom K, Solomon S (2000) Management of cluster headaches. In: Olesen J, Tfelt-Hansen P, Welch KMA (eds) The headaches, 2nd edn. Lippincott Williams & Wilkins, Philadelphia, pp 731–740
- Anthony M (1987) The role of the occipital nerve in unilateral headache. In: Rose FC (ed) Current problems in neurology: 4. Advances in headache research. John Libbey, London, pp 257–262
- 4. Ambrosini A, Vandenheede M, Rossi P, Alojd F, Sauli E, Pierelli F, Schoenen J (2005) Suboccipital injection with a mixture of rapid- and longacting steroids in cluster headache: a double-blind placebo-controlled study. Pain 118:92–96

- Torelli P, Manzoni GC (2004) Cluster headache: symptomatic treatment. Neurol Sci 25:S119–S122
- Peres MF, Stiles MA, Siow HC, Rozen TD, Young WB, Silberstein SD (2002) Greater occipital nerve blockade for cluster headache. Cephalalgia 22:520–522
- Busch V, Jakob W, Juergens T, Schulte-Mattler W, Kaube H, May A (2006) Functional connectivity between trigeminal and occipital nerves revealed by occipital nerve blockade and nociceptive blink reflexes. Cephalalgia 26:50–55
- Piovesan EJ, Kowacs PA, Oshinsky ML (2003) Convergence of cervical and trigeminal sensory afferents. Curr Pain Headache Rep 7:377–383
- 9. Goadsby PJ, Bahra A, May A (1999) Mechanisms of cluster headache. Cephalalgia 19[Suppl 23]:19–21

- Rozen TD (2005) Non-hypothalamic cluster headache: the role of the greater occipital nerve in cluster headache pathogenesis. J Headache Pain 6:149–151
- Afridi SK, Shields KG, Bhola R, Goadsby PJ (2006) Greater occipital nerve injection in primary headache syndromes – prolonged effects from a single injection. Pain 6: [Epub ahead of print]