Cephalalgia
An International Journal of Headache
International Headache Society

Cephalalgia
30(3) 365–367

DOI: 10.1111/j.1468-2982.2009.01882.x
cep.sagepub.com
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Does sphenopalatine endoscopic ganglion block have an effect in paroxysmal hemicrania? A case report

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Abstract

The authors report the case of a 69-year-old woman suffering from paroxysmal hemicrania (PH), intolerant to indomethacin and resistant to multiple therapies, in which sphenopalatine endoscopic ganglion block (SPG) dramatically modified the clinical outcome. SPG blockade could be considered a reasonable alternative in drug-resistant PH cases where indomethacin is contraindicated.

Keywords

Shenopalatine ganglion block, paroxysmal hemicrania

Date received: 17 November 2008; accepted: 1 March 2009

Paroxysmal hemicrania (PH) belongs to a group of headaches known as trigeminal autonomic cephalalgias (TACs), which are characterized by unilateral head and/or face pain with accompanying autonomic features (1,2). PH is characterized by severe, strictly unilateral pain attacks localized to orbital, supraorbital or temporal sites, or any combination of these, accompanied by one or more ipsilateral autonomic features. PH attacks are short (lasting 2–30 min) and are more frequent than cluster headache (CH). The hallmark of PH is the absolute cessation of pain with indomethacin, which distinguishes it from the other TACs (1,2). A 69-year-old nun suffering from PH, intolerant to indomethacin and resistant to multiple therapies, in which sphenopalatine endoscopic ganglion block (SEGB) dramatically modified the clinical outcome, is reported here.

The patient came to our Headache Centre complaining of headaches of increasing intensity occurring over a period of 13 months. Nothing particularly significant showed up in her medical history, and no recent trauma was referred. The patient had never suffered from headaches before, and there was no family history of headaches. The pain was described as severe, unilateral, localized to orbital and supraorbital areas, accompanied by ipsilateral autonomic features (rhinorrhoea, eyelid oedema, forehead and facial sweating and ptosis) lasting 5–20 min. The episodes had a frequency of 8–10 per day. Nocturnal episodes, although infrequent, were noted. In addition, no particular precipitating factors were reported.

Neurological examination was normal. Blood and urine tests, including coagulation parameters, were normal. Magnetic resonance (MR) imaging of the brain and MR angiography were both normal. Before coming to us, the patient had already been treated with amitriptyline, naproxen, diclofenac and ibuprofen, with no results. Once the diagnosis of PH was proposed, 50 mg indomethacin was administered three times a day, with dramatic response in terms of pain relief. In accordance with International Headache Society diagnostic criteria (2), the diagnosis of chronic PH was therefore confirmed. However, after a few weeks of therapy, the patient developed dyspepsia and nausea, and a gastroscopy revealed erosive gastritis, even though protein pump inhibitors were promptly started. Indomethacin was therefore stopped, and multiple treatments were tried, including celecoxib, piroxicam, topiramate (100 mg/day), verapamil (240 mg/day),

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lithium (900 mg/day), carbamazepine (800 mg/day) and gabapentin (900 mg/day), with no results. Block of the greater occipital nerve was also tried, but proved unsuccessful. We therefore proposed an SEGB, and the patient accordingly gave her consent.

SEGB is a new technique based on endoscopic ganglion blockade that approaches the pterygopalatine fossa through the lateral nasal wall, and consists of injecting a mixture of local anaesthetics and steroids as close as possible into the sphenopalatine ganglion (SPG). This technique, which was proposed in a recent study, has shown encouraging results in treating drug-resistant CH (3). Endoscopic control (30° rigid optics, 4 mm diameter) allows injection into the nasal mucous membrane immediately behind, and over, the middle turbinate tail, where the pterygopalatine fossa is deeply located. The needle is inserted into the inferior portion of the sphenopalatine foramen, and injected directly into the fossa. The procedure begins with nasal decongestion and topical local anaesthesia (2% mepivacaine with 1/100 000 adrenalin). A long 20-G needle is used to administer triamcinolone acetonide (40 mg), 1% bupivacaine (4 ml) and 2% mepivacaine with 1/100 000 adrenalin (2 ml). The procedure is repeated once a week for 5 weeks.

In the first 2 weeks, an important reduction in both frequency of episodes and intensity of pain was observed. At the end of treatment (after 5 weeks), the episodes had reduced to one a day and were treatable with 500 mg paracetamol, which provided complete relief. A follow-up of 4 months showed that the clinical improvement continued. The patient no longer needed to return for check-ups.

The pathophysiology of PH is poorly understood. Recent positron emission tomography studies have revealed, similar to CH (4), activation of the hypothalamus at the onset of episodes (ipsilateral activation to the pain side in CH, and contralateral in PH) (4,5). Dysfunction of the central control of the autonomic nervous system during PH and CH attacks has been documented, affecting both the sympathetic and parasympathetic systems (SPG and facial nerve) (6–9). It is therefore possible that PH and CH share the same pathogenesis (1,10).

A number of surgical approaches have been tried in chronic CH drug-resistant cases, and the SPG block seems to have some degree of efficacy (3,11,12).

There are no controlled trial options for the management of PH in patients where indomethacin is contraindicated. Topiramate has been reported to be useful (13,14), as well as sumatriptan (15) (the latter with conflicting results (16)). Grand occipital nerve injections with lidocaine and methylprednisolone are helpful in some patients with PH (17). Verapamil and acetylsalicylic acid may give partial relief (18), as well as piroxicam (19). Lastly, lithium, carbamazepine and other anticonvulsant drugs are mostly ineffective (20).

Our patient, who was drug resistant, showed a clear clinical improvement with an SEGB. The use of endoscopy certainly facilitates the identification of the anatomical region corresponding to the pterygopalatine fossa, thus making it possible to inject the target site directly. The SPG is located in the pterygopalatine fossa, behind the middle nasal turbinate under a 1–1.5-mm layer of connective tissue and mucous membrane, and in front of the pterygoid canal. In the absence of complex anatomical situations, this superficial location allows the block to be carried out (by injecting the drugs) with a reasonable degree of certainty, even though in our case no neurophysiological investigations were carried out to prove this. The duration of the anaesthetic block of mepivacaine and bupivacaine varies from 240 to 560 min, and co-administration of adrenalin slows the rate of systemic absorption of the anaesthetic, allowing the body more time to metabolize it and prolonging the block (21). The real duration of action of an anaesthetic, or the period during which it remains effective, is determined by its protein-binding activity, due to the fact that the anaesthetic receptors along the nerve cell membrane are proteins. The mechanism by which anaesthetic block alleviates pain is not fully understood. However, it is believed to reverse the parasympathetic contribution to intracranial vasodilation (22).

The cranial autonomic symptoms prominent in CH and PH may be due to central disinhibition of the trigeminovascular-autonomic reflex by the hypothalamus, possibly though direct hypotalamictrigeminal connections (10). It has been suggested that infiltrating the sphenopalatine region might block the indirect auto-feed nerve circuits leading to the hypothalamic centres from the SPG (3,9). However, additional and multifactorial aspects may also be involved.

Furthermore, we cannot exclude the fact that the improvement of the clinical course might be due to spontaneous conversion to the episodic form of PH. This is unlikely, however, because episodic forms typically progress into chronic PH, and the opposite is quite rare (23).

Therefore, taking the previous considerations into account, SPG blockade could be considered a reasonable alternative in drug-resistant PH cases where indomethacin is contraindicated.

Further controlled studies are advisable to confirm the efficacy of this approach in PH.

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