

## SHORT REPORT

## Secondary cluster headache responsive to sumatriptan

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### Abstract

**A patient was seen with secondary cluster headache whose acute pain responded promptly to sumatriptan. The headaches started after injury to the vertebral artery. This finding provides clinical affirmation of the existence of the trigeminal/cervical nuclear overlap that is central to this condition.**

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Cluster headache is a rare form of primary headache marked by unilateral excruciating pain in association with autonomic features.<sup>1</sup> It is unusual to see as a secondary phenomenon and in this setting its management is a considerable challenge. A case of secondary cluster headache is presented which has allowed the chance to examine the utility of sumatriptan in this clinical setting.

### Case report

The patient, a 67 year old man, presented with a three and a half year history of episodic headache. The headaches started after a fall from a hospital bed during which he jammed his head between the bed and the bedside locker. He experienced severe neck pain and vertigo on upward gaze, and was found on cerebral angiography to have dissection of the right vertebral artery. Within two days of the injury, his episodic headaches began. After this he had several bouts of headache punctuated by periods completely headache free. His pain typically started in the neck and occipital area and radiated to the left temporal region to become a very severe boring pain. The headaches were accompanied by watering of the left eye, blockage of the left nostril, and a Horner's syndrome on the left (witnessed by PDC during an attack). The attacks last between 30 minutes and 12 hours but are usually a few hours. He may experience up to five attacks a day during an active bout. Red wine would precipitate an attack.

The first bout of headache lasted for three months. At the initial recurrence he was treated by injection of the left greater occipi-

tal nerve with local anaesthetic which left him pain free for several days. After the headaches returned he was treated with a C2 facet block which left him without headache for six months. The headaches subsequently recurred for another six months before being controlled by methysergide. For acute attacks, oxygen treatment often worked well if used early enough. Oral ergots were of no use. During the hospital admission, he was given sumatriptan (6 mg subcutaneously), which aborted the attack very rapidly, within minutes. This approach was used several times and on each occasion his headache responded rapidly to sumatriptan. He had no side effects aside from some irritation at the site of injection. He was started on valproate (500 mg twice daily), which brought his headaches under control.

Physical examination was mostly unremarkable. The cranial nerves were normal. In particular there was no evidence of trigeminal sensory loss or Horner's syndrome between attacks of headache. Horner's syndrome was seen during an acute headache. He had no other relevant clinical signs.

### Discussion

#### SECONDARY CLUSTER HEADACHE

We present a case of the uncommon condition of secondary episodic cluster headache. It has not been previously reported after vertebral artery dissection. The headaches show many typical features of cluster headache, including ipsilateral lacrimation, rhinorrhoea, and Horner's syndrome. The unilateral boring pain and the temporal spacing of attacks are also typical, as is the response to oxygen, and the initial response to methysergide.<sup>2</sup> The duration of the pain is perhaps a little long and the patient is older than average for the usual idiopathic cluster headache.

There have been only a few reports of secondary cluster headache. In a series of 11 patients, Matthew and Rueveni noted that most cases followed facial trauma with soft tissue injury.<sup>3</sup> In all but one patient, injuries occurred in the trigeminal territory. Although the attacks looked like cluster headache, with ipsilateral autonomic features, they did not show the usual periodicity or remissions and were refractory to treatment. Cluster headaches have also been reported with an

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arteriovenous malformation in the occipital lobe,<sup>4</sup> pituitary adenoma,<sup>5</sup> and upper cervical meningioma.<sup>6</sup> Of particular interest in regard to our case is a report of a 55 year old man with a 12 year history of cluster headache on the right side associated with an ipsilateral vertebral artery aneurysm.<sup>7</sup> The headaches resolved when the aneurysm was clipped. The authors postulated that the aneurysm irritated the C1 and C2 fibres that innervate the dura mater. Koenigsberg *et al*<sup>8</sup> reported cluster headaches developing in association with a pseudoaneurysm of the intracavernous carotid artery. Their patient supports the generally held view that the cavernous sinus is a key locus of pathology in cluster headaches, with a convergence of fibres from the first two divisions of the trigeminal nerve as well as both sympathetic and parasympathetic fibres. The patient presented here suggests a more complex central mechanism for the initiation of attacks.

#### RESPONSE TO SUMATRIPTAN

The patient in this study responded promptly to sumatriptan. To our knowledge sumatriptan has not been previously used in secondary cluster headache. Sumatriptan is a 5-hydroxytryptamine (5HT<sub>1D</sub>) receptor agonist, which has been shown to be effective in randomised, double blind, placebo controlled trials in patients with primary cluster headache.<sup>9</sup> The rapidity of action and consistent responses reported anticipate the responses reported here.<sup>2</sup> This consistent and rapid onset was of particular importance in light of the relatively long headaches experienced by our patient when untreated. It has been shown recently that acute attacks of cluster headache are associated with activation of the trigemino-

vascular system as reflected by increases in neuropeptide markers of this system that rapidly return to normal after treatment with sumatriptan.<sup>10</sup>

In summary, we have seen a patient with secondary cluster headache whose acute pain responds promptly to sumatriptan. The patient's headaches started after injury to a posterior circulation vessel, the vertebral artery. This finding, together with other reports, provides clinical affirmation of the existence of the trigeminal/cervical nuclear overlap that is central to understanding this condition. It can be suggested, albeit with caution as this is an isolated case, that sumatriptan acts on the final common pathway of the expression of the cluster headache syndrome rather than on the fundamental defect generating the episodes.

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