

LETTER TO THE EDITOR

DEPENDING ON THE UNDERLYING PATHOPHYSIOLOGY, HEADACHE IN SUBARACHNOID HEMORRHAGE MAY NOT ALWAYS RESPOND TO BLOCKING OF THE OCCIPITAL NERVES

Dear Editor:

We read with interest the article by Gogoi et al. regarding a 35-year-old woman who suffered a subarachnoid hemorrhage (SAH) following the rupture of an aneurysm of the right internal carotid artery (1). She was treated with coiling, but the course was complicated by hydrocephalus and severe headaches (1). The hydrocephalus was managed via the implantation of a ventriculoperitoneal shunt, while the headaches were treated with bilateral greater occipital nerve blocks, resulting in a 50% reduction in pain (1). The authors concluded that there is evidence suggesting peripheral nerve blockade may be an effective method for pain management in SAH patients (1). While the study is insightful, it warrants further discussion.

A primary point is that headaches in SAB patients do not necessarily stem from a single pathophysiological mechanism but can arise from various mechanisms. As one of the cardinal symptoms of SAB, headaches can develop through at least three distinct mechanisms. When blood flows into the subarachnoid space and mixes with cerebrospinal fluid, intracranial pressure can rise. Furthermore, clotted blood, disintegrating cells, and iron can irritate the sensitive pain fibers of the meninges, thereby stimulating pain receptors. Headaches can also result from arterial spasms and inflammation caused by blood breakdown products. These trigger local inflammation and subsequently lead to the narrowing of cerebral arteries (2). Given these considerations, it is understandable that different pathophysiological mechanisms may require different approaches for the specific treatment of headaches associated with SAB.

A second point is that the migraine diagnosis for the index patient is questionable (1). Although the headaches were described as unilateral, their character was not pulsating, as is typical for migraine headaches. Furthermore, no hypersensitivity to light or noise was

reported either at the onset or during the attacks. It is also surprising that the patient never received triptans during headache attacks as a response to triptans could have indicated that the preceding headaches were indeed migraines. If migraine was indeed the correct diagnosis, it would need to be clarified why the patient did not receive GLP-1 receptor agonists for prophylaxis and to reduce headache frequency.

A third point is that the study design (case report) is unsuitable for drawing general conclusions about the efficacy of a drug or intervention (3). Case reports lack scientific robustness and they provide no basis for extrapolating findings to a broader population. It is quite likely that the researchers' personal views influence the case study, leading to researcher bias. Since case reports are not based on representative samples, they provide no information on rates, ratios, indices, or prevalence. Moreover, case reports are susceptible to publication bias (3). It should also be noted that the report on the index patient did not comply with the CARE guidelines. The CARE guidelines are designed to enhance the completeness, accuracy, and transparency of published case reports.

A fourth point is that consideration was not given to the possibility that the index patient's headaches might have been attributable not only to the SAB but also to increased tension in the neck muscles. This could explain why the occipital nerve block proved beneficial for the index patient.

Until the efficacy of nerve blocks for treating headaches in SAB patients is confirmed by double-blind, placebo-controlled crossover studies, it should not be recommended as an evidence-based treatment method.

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