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Post-Spinal Headache: A New Possible Pathophysiology

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Dear Editor,

Postdural puncture headache (PDPH) remains a major concern after spinal anesthesia and is known to complicate 0.5% - 24% of cases (1). This complication is believed to be due to a reduction in brain pressure, caused by the leakage of cerebrospinal fluid (CSF) (2). A decline in CSF pressure leads to traction on pain-sensitive parietal dura and intracranial structures, thereby causing subsequent headache in patients (3, 4). In these cases, the rate of CSF loss through dural perforation is greater than the rate of CSF production, especially when the needle size is larger than 25G (4).

Gadolinium-enhanced magnetic resonance imaging (MRI) in PDPH frequently indicates the sagging of intracranial structures (4). MRI may also signify meningeal enhancement, which could be due to vasodilatation of thin-walled vessels in response to intracranial hypotension (4). Although finer needles have been used in recent years, the incidence of PDPH has not significantly decreased (5). In this regard, Grant et al. by using MRI showed a major decline in intracranial CSF volume after lumbar puncture, which was frequently associated with PDPH; however, some patients developed PDPH with relatively little alterations in the intracranial CSF volume (4).

The 30% - 60% efficacy of blood patch technique, as the gold standard for the treatment of PDPH, confirms the theory of CSF leakage, while some other documented treatments for PDPH suggest other pathophysiological explanations for PDPH (6). We postulate that brain hyperperfusion due to the sudden increase in cerebral blood flow (CBF) after sudden global vasodilation in spinal anesthesia might be one of the underlying mechanisms of PDPH. Hyperperfusion syndrome, a clinical syndrome presenting as a migrainous phenomenon, transient focal seizure activity, or intracerebral hemorrhage following carotid endarterectomy, was initially described by Sundt in 1981 (6).

In addition, impaired cerebral autoregulation seems to play a significant role. The brain is able to maintain constant intracranial pressure through its autoregulatory mechanisms when an alteration in blood flow occurs. The low-flow state, induced by hypotension after spinal anesthesia, can result in the compensatory dilation of cerebral vessels to maintain adequate CBF. This dilation might cause the vessels to lose their ability to autoregulate vascular resistance in response to the sudden increase in blood pressure. As a consequence, CBF increases after the rise in blood pressure, which can cause headaches, similar to what occurs in hyperperfusion syndrome (4). This mechanism has been also suggested for explaining postictal agitation and headaches, associated with electroconvulsive treatments (4).

Rizvi et al. have successfully used mannitol infusions in PDPH treatment for years (3). Mannitol acts by the following mechanism: An acute rise in blood osmolality decreases the brain water content, thereby decreasing the brain bulk and intracranial pressure and increasing the intracranial compliance (1). Researchers believe that refloatation of the brain is the main mechanism of mannitol for alleviating PDPH (1). However, we assume that the reduction in brain bulk by mannitol prevents hyperperfusion, which occurs with the augmentation of blood pressure after the sudden decline in blood pressure following spinal anesthesia.

Tazeh-Kand showed that administration of 5 ml of normal saline before the intrathecal administration of hyperbaric bupivacaine, as a preventive approach, can be an effective way to minimize PDPH in patients undergoing cesarean section (6). The explanation may be as follows: Intrathecal administration of normal saline (5 mL) increases CSF pressure and through this mechanism prevents brain venodilatation and hyperperfusion after recovery from spinal anesthesia. Caffeine has been also frequently used for the management of PDPH (7). In fact, caffeine induces cerebral vasoconstriction by antagonizing adenosine and by this mechanism prevents brain hyperperfusion and alleviates headache (8).

Overall, our theory reflects the Monro-Kellie doctrine

(7), which indicates that the sum of brain volume, CSF, and intracranial blood is kept constant. CSF loss results in an increase in blood volume due to venodilatation, which might be the cause of headache.

Footnotes

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