EDITORIAL

Augmenting Hypertensive Therapy in Patients with Postoperative Subarachnoid Hemorrhage: What's the Right Choice?

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Contrary to the historical practice of "Triple H" in the management of postoperative patients of subarachnoid hemorrhage (SAH), in the present times, augmented hypertensive therapy is the mainstay in improving cerebral perfusion pressure.[1] Most practitioners use vasopressors such as norepinephrine to augment mean arterial pressure (MAP) in a stepwise fashion (20–30%) until symptoms improve or a MAP of 120–130 mm Hg is reached. No single vasopressor is found superior.[2]

We know that induced hypertension increases cerebral blood flow (CBF) and brain tissue oxygenation and reverses vasospasm-related neurological deficits.[3] Researchers have also suggested ionotropic agents improve CBF and reverse neurological deficits in patients who do not respond to vasopressors.[4]

Studies have demonstrated that vasoconstrictors do not directly affect cerebral hemodynamics in health.[5] However, their effectiveness in patients with SAH has not been studied. In an interesting study conducted by Lakshmegowda et al.[6] published in this issue of the journal, the authors hypothesized that in patients with SAH, vasopressor administration increases the CBF velocity, depending on the intactness of the CBF regulatory mechanisms. The authors determined that blood pressure augmentation with norepinephrine infusion in postoperative aneurysmal subarachnoid hemorrhage patients increases CBF velocity on the cerebral hemisphere with impaired autoregulation, an effect that is desirable in patients with focal cerebral ischemia. They also found that these CBF velocity changes happen irrespective of the cardiac output changes following norepinephrine infusion. The authors have shown a differential effect of hypertensive therapy related to the intactness or impairment of CBF autoregulation.

In this context, one of the first randomized trials that investigated whether induced hypertension improved the outcome of patients was the Hypertension Induction in the Management of AneurysmaL subArachnoid haemorrhage with secondary IschaemiA (HIMALAIA) trial, which was stopped early due to slow enrollment of patients. It was unable to show the clear benefits of blood pressure augmentation for functional outcomes.[7] In a recent study on 98 patients, Steiger and colleagues assessed the hemodynamic response and clinical outcome following intravenous milrinone plus norepinephrine-based hyperdynamic hypertensive therapy in patients suffering secondary cerebral ischemia after aneurysmal SAH.[8] The authors demonstrated improved cerebral perfusion by the milrinone and norepinephrine-based hyperdynamic therapy.

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The study by Lakshmegowda et al. suggests hypertensive therapy with norepinephrine increases CBF only in regions with impaired autoregulation, an effect that is desirable in patients with focal cerebral ischemia. At the same time, it does not affect normal regions of the brain. While the findings of this study are definitely important and provide ground for future research, the limitations of this study cannot be overlooked. Some of them have already been mentioned in their article. An important limitation is the lack of data on the long-term outcome. It would have been meaningful, clinically, if the authors provided information on the outcome of their patients.

I agree with the authors' conclusion that further studies are required to study the mechanism of changes in cerebral hemodynamics with various vasoactive drugs using direct CBF measurement methods. The results of such studies will help clinicians to choose the appropriate agent for hypertensive therapy in patients with aneurysmal SAH.

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