Commentary: Posterior Reversible Encephalopathy Syndrome as a Complication of Induced Hypertension in Subarachnoid Hemorrhage: A Case-Control Study

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In this study, the authors describe the incidence of Posterior Reversible Encephalopathy Syndrome (PRES) in a cohort of patients with subarachnoid hemorrhage (SAH) who underwent induced hypertension (IH) therapy for delayed cerebral ischemia (DCI).1 In their management protocol, DCI is treated, first with fluids administration, then by vasopressor infusion, with a targeted mean arterial pressure (MAP) adjusted to the clinical response. Median duration of IH was 8 (4-15) d in patients with PRES versus 5 (2-9) in patients without. The authors interestingly reported 7% of PRES and found that a targeted MAP > 135 mmHg or > 50 mmHg compared to baseline values, where predictive of PRES occurrence. Despite potential bias due to the observational retrospective design of the study, this article raises some important aspects to discuss in the management of DCI after SAH.

The complex management of patients with SAH and the potential systemic (ie, acute cardiac dysfunction, neurological pulmonary edema) or neurologic (ie, recurrence of bleeding, DCI) severe adverse events may largely justify patients’ admission in Neuro-ICU, where they can benefit of a continuous surveillance and monitoring.2,3 The choice of the monitoring tools is of paramount importance in order to detect these events, to evaluate the effects of the therapies undertaken as well as potential undesirable effects (ie, acute cardiac failure, PRES, acute renal failure). In this study, the authors use MAP as the corner stone of the management of patient with suspected ICD. They hypothesize that an increase in MAP leads to an increase in cerebral blood flow, especially in areas of vasospasm. Some commentaries are valuable on this point.

Recent data question the interest of IH therapy in the management of DCI, indeed, Gathier et al4 did not found a significant difference in CBF with computed tomographic perfusion in patient treated with IH therapy for DCI compared to control group. Furthermore, in a recent randomized controlled trial, prematurely stopped for lack of effect and inclusion difficulties, the authors observed no effect of IH therapy on neurological outcome.5

From a pathophysiological point of view, in the absence of autoregulation (or if MAP exceeds the limits of autoregulation), MAP raising increases the CBF. If cerebral autoregulation is preserved, the increase in MAP on the autoregulating plateau is not accompanied by an increase in cerebral blood flow.6 In these 2 cases, the MAP increase by the use of vasopressor, due to the excessive increase of the left ventricular afterload, may lead to a decrease in cardiac output (CO) that may contribute to a decrease in CBF.7

Indeed, it is also known that using phenylephrine can lead to a CO decrease, compared to norepinephrine.8,9 In a retrospective study, patients treated with phenylephrine for DCI after SAH were less responder to IH treatment in terms of clinical improvement and had more delayed infarct on imaging, compared to patients treated with norepinephrine.10 In this direction, interesting results showed an increase in CBF secondary to an increase of CO in patients with DCI, independently of MAP.11

Thus, if the choice of the vasopressor is of importance, probably more important is the choice of the most appropriate monitoring devices, in regard of the patient condition and the choice of the targets. In the past decades, the major importance of CO monitoring to guide the fluid management has been highlighted.
in many critical conditions, in ICU as well as in operating room. Neurological patients may present hemodynamic instability that would justify CO monitoring. In the case of DCI, CO seems to represent an interesting target to improve neurological patient outcomes, justifying monitoring CO whatever the hemodynamic status.

If systemic monitoring is essential for these patients, neurological monitoring (including transcranial Doppler [TCD], jugular venous saturation, or invasive intracranial pressure measurement) is also crucial to evaluate the effects of the treatments. The choice must be adapted to the clinical state and neurological status evolution. In this study, patients did not have TCD monitoring. This choice is debatable, as cerebral vasculature evaluation by TCD can reveal simple and reliable to assess the response to the therapeutics, and also help to define therapeutic targets, as MAP and CO, especially in patients under neuroprotective sedation or comatose patients. In the study, IH therapy was continued for relatively long duration, with clinical evaluation as the only criterion of evaluation of IH therapy efficiency. A multimodal monitoring could permit to target lower MAP, and/or shorter duration of IH therapy and maybe to avoid PRES.

This article shows that MAP guided IH therapy presents significant risks of adverse events. Together, these elements call for the importance of a global, multimodal, systemic as neurological monitoring, which could allow regular evaluations to define therapeutic targets in a multidaily manner, and thus avoid excessive and potentially deleterious therapeutics as PRES induce by IH therapy. To validate this attitude, randomized controlled trials are needed to compare a patient tailored management to a management only based on MAP in patients with SAH.

Disclosure
The authors have no personal, financial, or institutional interest in any of the drugs, materials, or devices described in this article.

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