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Intracranial pressure after aneurysmal subarachnoid hemorrhage - Time to revisit

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Long since the days of pneumoencephalography, it has been known that massive aneurysmal subarachnoid hemorrhage (aSAH) may result in significant elevation in intracranial pressure (ICP) which can be fatal without prompt treatment and can trigger a vicious cycle of low cerebral perfusion induced ischemia in turn causing further ICP elevation [1]. Management of aSAH has evolved significantly during recent times with increasing number of patients, especially poor-grade patients receiving endovascular treatment [2]. Surgical and endovascular treatments of cerebral aneurysms create significantly different pathophysiologic milieu in the cranial vault [3], and therefore, call for a need to revisit.

In this issue of *Critical Care Medicine* Zoerle et al. studied incidence and predictors of elevated ICP in aSAH and its association with 6-month outcomes [4]. The authors analyzed 116 cases treated in a single academic center from a prospective observational database. Of note, almost three quarters of patients were treated with endovascular coiling. The patient cohort had high severity with 80% patients classified as World Federation of Neurosurgical Surgeons (WFNS) grade IV–V and 98% classified as Fisher grade 3–4. Among this cohort, 36% of patients had a maximum mean ICP >20 mm Hg (calculated over 12 hour intervals), the frequency of which peaked on day 3 after the ictus. Factors independently associated with highest mean ICP >20 mmHg were pre-treatment neurological status (WFNS grade), rehemorrhage, early lesions on CT and worse Fisher grade. In multivariate analysis, highest mean ICP was the only factor associated with 6-month mortality with most deaths occurring in the first 7 days. However, consistent with a study from Heuer et al [5], ICP was not independently associated with unfavorable 6-month outcomes. Neurological status, age and CT lesions at any time were significant independent predictors of 6-month outcomes in regression analysis.

It is highly significant therefore, that in a population of severe aSAH patients requiring ICP monitoring for either severe coma or acute hydrocephalus, elevated ICP was the most

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important factor determining mortality. This study does not tell us which patients should be monitored, but it does support the need to monitor ICP in high-grade aSAH and to treat ICP aggressively. An important assumption made was that all ICP episodes were maximally treated minimizing the duration of such events. The therapy intensity level results for management of intracranial hypertension in this study indicated that high intensity treatment was significantly more often utilized in patients who died or had unfavorable outcomes vs. survivors and those with good outcomes. If therapy intensity was applied appropriately, then persistent elevated ICP may represent refractory cases for which increased cerebrospinal fluid (CSF) drainage (93% had external ventricular drains) and other therapies were less beneficial.

As for long-term functional outcomes, it is hardly surprising that in a complex disease such as aSAH, elevated ICP was not an independent predictor. As noted by Zoerle and others [6], factors such as global cerebral edema from ictal cerebral circulatory arrest, neurotoxicity of blood and its degradation products, disruption of cerebral autoregulation and cerebral vasospasm may significantly contribute to brain injury and poor outcomes after aSAH, and are far less amenable to reliable monitoring and target based therapy.

Now we may ask whether we have learned anything new. In 1982, Voldby recorded intraventricular pressure in 52 aSAH patients for an average of 8 days [7]. A close correlation was found between changes in clinical grade and in mean ICP. Drainage improved the condition in uncomplicated cases, but was less effective or ineffective when severe vasospasm or rebleeding occurred. Patients with severe spasm had permanently elevated ICP, and mean ICP exceeding 25 mm Hg for the whole monitoring period was associated with a poor prognosis. This study recommended that patients with even mild neurologic impairment (Hunt and Hess grades II–III) be considered for ICP monitoring to prevent deterioration and that those with severe grades (IV–V) be monitored due to the high prevalence of intracranial hypertension. In 2004, Heuer et al [5] analyzed 433 patients with surgically treated aSAH and found that elevated mean hourly ICP >20 mm Hg occurred in 54% of cases. Almost half of patients with good clinical grade (Hunt and Hess Grades I–III) also had elevated ICP. Failure of increased ICP to respond to treatment was always associated with poor outcome, although many patients whose elevated ICP could be controlled had a satisfactory outcome. The current study adds the following new or important findings: (1) mode of aneurysm treatment (surgical clipping vs. endovascular coiling) does not appear to be a significant factor in occurrence of high ICP; (2) hydrocephalus on initial imaging is not significantly associated with high ICP, likely due to the high prevalence of EVD placement and CSF drainage in this study; and (3) the independent association of early lesions on CT and worse Fisher grade with high ICP support previous studies suggesting that high ICP can worsen vasospasm, and that ICP reduction, in particular osmotic diuresis may reverse vasospasm development and clinical signs of delayed ischemia [8, 9].

American Heart Association guidelines recommend that aSAH-associated acute symptomatic hydrocephalus should be managed by cerebrospinal fluid diversion [10]. The role of ICP monitoring in the absence of hydrocephalus has not been established for this condition. Results of this and prior studies would indicate that poor-grade patients with

aSAH should receive ICP monitoring during the acute phase either with an external ventricular drain with careful CSF drainage or with an intraparenchymal ICP monitoring device. Although good-grade patients were less represented in this study, the presence of high Fisher grade may also be a reasonable indication for ICP monitoring.

This report adds to a limited number of studies assessing the course of ICP, predictive factors and impact on outcomes in patients with aSAH. The results are generalizable to mainly high-grade aSAH and patients treated with endovascular coiling. Although cases of truly refractory ICP may have limited options, improving cerebral perfusion by lowering increased ICP remains a reasonable treatment against delayed cerebral ischemia considering the inadequacy of our medical management thus far.

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