CRITICAL CARE

COMMENTARY

Wet lungs, broken hearts and difficult therapies after subarachnoid hemorrhage

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See related research by Hoff *et al.*, http://ccforum.com/content/14/2/R43

Abstract

Pulmonary edema (PE) can occur after subarachnoid hemorrhage and can jeopardize arterial oxygenation, which is essential for a suffering brain. In some cases PE is evident in the emergency room, being the direct consequence of intracranial bleeding, which causes an immediate and overwhelming catecholamine discharge. In the following days, PE can occur because of cardiac failure, often related to initial cardiac damage, concurrent therapies with fluid overload and vasopressors, infections, or pre-existing co-morbidities. The causes of PE need to be identified for appropriate treatment.

An acute catecholamine discharge immediately following intracranial bleeding can have devastating extracranial effects: the heart, lungs, fluids and electrolytes can be severely affected. Hoff and colleagues report on pulmonary edema (PE) following subarachnoid hemorrhage (SAH) [1]. They found a high incidence of PE, usually several days after the initial bleeding, and an association of PE with lower intravascular volumes (compared with cases without PE). PE is not uncommon after SAH, both as an early complication and as a late complication.

Neurogenic PE is an acute event directly linked with an intracranial disaster; as such, it is often diagnosed on admission in cases with severe SAH [2]. PE in the days following SAH has been reported in association with triple-H therapy, which includes hypervolemia and induced arterial hypertension [3]. In different series, the PE incidence varied from 14 to 23% [4,5]. Increasing evidence is accumulating on the myocardial dysfunction that follows SAH, as well as other acute intracranial disasters. Takotsubo syndrome – with typical left ventricular abnormalities (bulging out of the apex of the heart with preserved function of the base, which earned the syndrome its name – a kind of a pot used as octopus trap in Japan), ECG abnormalities and biomarker changes – is a typical example of how much an injured brain can break the heart [6]. Neurogenic in origin, left ventricular failure causes congestion in the pulmonary vasculature and PE in Takotsubo syndrome.

What is concerning in the report of Hoff and colleagues is that PE, often labeled in the text as neurogenic PE, has been diagnosed days after the initial bleeding, without concurrent reduction of the cardiac index and in the absence of volume expansion [1]. The clinical implications of these findings, as pointed out by the authors, would be that PE more than 4 days after SAH is not cardiogenic and would not be suitable for treatment with diuretics. It is therefore hard to reconcile the findings of this paper with current knowledge.

Perhaps the methodology and terminology used in Hoff and colleagues' paper require further attention. PE has been identified by clinical signs and bilateral pulmonary infiltrates on the chest X-ray scan, without quantitative measures concerning the cardiac and respiratory function at the time of PE diagnosis. It is surprising that no data on oxygenation, such as a simple blood gas analysis, are reported. The hemodynamic status seems better documented, since the cardiac index and the circulating blood volume were estimated. There are two weaknesses in these data, however, the first of which is the reliance on a single technique without confirmation by independent measures. The second, and more relevant, weakness concerns the data used in this specific analysis: the data do not represent the actual circulating blood volume and cardiac index at the time of PE. In fact, mean values calculated from day 1 to the day when PE developed have been entered in the analysis. It becomes almost impossible, therefore, to use these mean data for correctly understanding what caused PE. Interestingly, diuretics were used in 65% of patients with PE in the days before the PE diagnosis. Were they used for correcting an

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excessive volume expansion, as seems likely? If that was the case, PE could have been cardiogenic, and/or somehow related to fluid management, without necessarily depending on a neurogenic cause.

Managing patients after severe SAH is challenging: on one hand, their brain requires normal (or even high, in the case of vasospasm) arterial pressure to warrant cerebral perfusion and prevent delayed ischemic deficits; on the other hand, their heart may be damaged by an early sympathetic discharge, causing ischemic damage, and does not tolerate an increased workload. PE is a deleterious complication that may worsen systemic and cerebral oxygenation, and as such needs to be quickly recognized and treated [7]. The enthusiasm for triple-H therapy, which carries the risk of fluid overload and an indiscriminate use of vasopressors, must therefore be tempered.

Perhaps the most important lesson to be drawn from this paper [1] is the need for accurate monitoring, both cardiovascular and respiratory, in all SAH cases. Some patients may simply require good clinical surveillance and careful fluid balance. The more severe cases, on the contrary, should be cared for by a team capable of detecting early signs of heart failure, identifying the causes of PE and treating them, still preserving cerebral perfusion.

Abbreviations

PE, pulmonary edema; SAH, subarachnoid hemorrhage.

Competing interests

The author declares that they have no competing interests.

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