

# **Surgical Neurology International**

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Letter to the Editor

# Letter to the Editor regarding "Comparison of postoperative cognitive dysfunction with the use of propofol versus desflurane in patients undergoing surgery for clipping of aneurysm after subarachnoid hemorrhage," which is the right time to evaluate?

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#### To the editor

Sharma et al. published a very interesting prospective double-blind study, in which they compared the influence of two anesthetics, frequently used, on the development of postoperative cognitive deficit (POCD) in patients undergoing surgical clipping of aneurysm due to aneurysmal subarachnoid hemorrhage (aSAH). The authors reported an incidence of POCD of 82% in the group treated with desflurane and 65% in those treated with propofol using the MOCA system. The authors conclude that although there was no significant difference between groups, there was a significant difference in the average scores of certain domains of cognitive function. [9]

We agree with the authors when they conclude that POCD is very common in survivors of aSAH, a condition that seriously compromises their long-term quality of life. POCD is considered to be the most common long-term neurological complication in patients with aSAH.<sup>[7]</sup>

However, in the study, the researchers only took into account the type of anesthesia to explain the presence of POCD within the first 2 weeks of bleeding, a period in which other complications of the same aSAH, namely cerebral vasospasm, delayed cerebral ischemia (DCI), and hydrocephalus can significantly compromise the neurological status of the patient and can significantly influence the development of POCD.

Previous studies confirm that in the context of aSAH, both hydrocephalus and vasospasm (or a consequent late ischemic déficit) are some of the factors that affect the development of cognitive deficit (CD) after aSAH in the short and long term. [3,6,7,10]

Vasospasm can occur acutely in the intraoperative and immediate postoperative periods due to manipulation of the vessels by the surgeon and from day 3 to day 21 postbleeding with a peak around day 7 and is described as the main cause of morbidity and mortality in patients who survive more than 2 weeks after aSAH generating a high risk of DCI.<sup>[5]</sup> Hydrocephalus, on the

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other hand, can appear from early postbleeding phases and in up to 10-46% of patients chronically generating neurological deficit and CD.[2,3]

The studies by Nordenmark et al. and Hütter et al. evaluated the factors associated with early CD (on average at the 1st 11 days after aSAH in the first study and 5.9 days after aSAH in the second), observing that the presence of stroke, Fisher scale Grades 3 and 4 and hydrocephalus secondary to a SAH were the main factors associated with CD.[3,7]

Despite recent advances in the acute management of aneurysmal hemorrhages, aSAH remains associated with mortality and poor functional outcome. The high mortality rate after successful management of an acute aneurysm is mainly attributed to DCI. Although 20-40% of patients with aSAH, who survive the initial hemorrhage, develop this phase of brain injury, which is characterized by a reduced level of consciousness and focal neurological deficits, mainly between days 4 and 14 after the initial bleed.[1]

Evidence from clinical and animal trails suggests that the presence of cerebral vasospasm is not a prerequisite for DCI, and therefore, DCI has a more multifactorial etiology.

On the other hand, hydrocephalus is a common complication of aSAH. Although 15-20% of patients with aSAH have acute hydrocephalus. Acute hydrocephalus after aSAH is associated with an altered level of consciousness. This finding is more frequent in patients with Hunt and Hess grades higher than III. The prognosis is poorer than in those patients with aSAH and without hydrocephalus. The incidence in an unselected series of patients is between 6.5% and 67%.<sup>[4]</sup>

The incidence of requiring a ventricular shunt is between 10% and 20% of cases. The pathogenesis of shunt-dependent hydrocephalus involves obstruction by blood products or adhesions that block the circulation of cerebrospinal fluid (CSF) within the ventricular system and aseptic inflammation after aSAH that can cause malabsorption of CSF. Furthermore, alterations in CSF dynamics can also affect CSF circulation and absorption.

Although the occurrence of hydrocephalus after aSAH has been well studied and some measures have been applied to prevent hydrocephalus, its possible risk factors remain unclear.[8]

Sharma et al. did not consider evaluating the presence of vasospasm or hydrocephalus in their patients. In addition, the authors chose to evaluate POCD in the critical period of presentation of neurological complications secondary to aSAH. It is very interesting to study the effect of anesthetics on POCD, but at least in the context of the patient with a aSAH, we consider that these complications should be taken into account in the statistical analysis of studies evaluating early CD after aSAH. The effect of vasospasm, DCI, and

hydrocephalus on this point is likely to be high, making it difficult to attribute the findings to the type of anesthesia

We believe that CD in this group of patients is mainly caused by the aSAH itself and its multiple possible complications than merely by the surgery and anesthesia used. [3,6,7,10] It is very likely that in a seriously ill patient the type and quality of anesthesia and surgery performed to influence the final cognitive result, but the pathophysiological weight seems to be much higher due to the underlying disease, for what we think that more than a POCD, the CD is more a neurological deficit due to the aSAH itself.

We think that the time chosen by the authors to study POCD after aneurysm clipping surgery in patients with aSAH does not clearly represent the effect of the type of anesthesia used.

#### Declaration of patient consent

Patient' consent not required as there are no patients in this study.

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#### **Conflicts of interest**

There are no conflicts of interest.

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