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Postoperative ICU management of patients after subarachnoid hemorrhage

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Abstract

Purpose of review—This article reviews recent advances in the postoperative ICU management of patients after subarachnoid hemorrhage (SAH), especially with regards to hemodynamic management, methods of improving neurological outcomes, and management of cardiac and pulmonary complications.

Recent findings—Several hemodynamic monitors and parameters may be useful for guiding volume therapy, including cardiac output, stroke volume variation monitoring, and global end-diastolic volume index. Early goal-directed hemodynamic therapy after SAH has recently been shown to improve clinical outcomes in patients with a poor clinical grade or coexisting cardiopulmonary complications. Recent laboratory and imaging modalities are being developed to identify patients at risk for developing vasospasm after SAH. Evidence for the use of various prophylactic adjuvant therapies to prevent vasospasm, including magnesium, phosphodiesterase 3 inhibitors, and therapeutic hypothermia, is emerging. Intrathecal administration of vasodilators or fibrinolytics may have offered advantages over systemic drug administration in the treatment of vasospasm. Pulmonary and cardiac complications are common after SAH, and are associated with an increased risk of mortality.

Summary—The postoperative ICU period after SAH is associated with a significant morbidity and mortality risk, and recent studies have greatly contributed to our understanding of how to optimally manage these patients.

Keywords

cerebral vasospasm; delayed cerebral ischemia; ICU management; subarachnoid hemorrhage

Conflicts of interest

There are no conflicts of interest.

INTRODUCTION

The diagnosis and management of acute subarachnoid hemorrhage (SAH) represents a major challenge for practitioners. Although SAH accounts for only 5% of strokes, the disease often presents in patients younger than 55 and is often fatal [1]. Up to 15% of patients with SAH die before reaching the hospital, and the overall mortality rate of SAH approaches 50%. After initial treatment with endovascular coiling or direct surgical clipping, patients are typically managed in the ICU.

Cerebral vasospasm and delayed cerebral ischemia (DCI) are major complications of SAH, and early postoperative care includes the administration of oral nimodipine and maintenance of cerebral blood flow (CBF). The course of acute SAH is often complicated by pulmonary and cardiac dysfunction. Although great strides have been made in the last decade in decreasing mortality and improving neurological outcomes after SAH, postoperative management remains a challenge.

In recent years, there has been much interest in postoperative hemodynamic management, methods of improving neurological outcomes, and identifying and treating other non-neurological complications of SAH. In this article, we will highlight some of the recent studies that have contributed to our understanding of how to best manage patients in the early period after SAH.

HEMODYNAMIC MANAGEMENT

Optimal hemodynamic management is essential in the early period after SAH. Patients with SAH are prone to hemodynamic instability that may result from impairment of cerebral blood flow autoregulation and cardiac dysfunction. Volume resuscitation should be aimed to provide adequate preload to maintain cardiac stability and optimal CBF and oxygenation. Hemodynamic insufficiency related to hypovolemia or low cardiac output is an important cause of secondary brain injury and DCI, which are associated with an increased risk of death and poor neurological outcomes. Avoiding volume overload and pulmonary edema is equally important, and may be particularly challenging in patients with a poor grade SAH in whom hypovolemia and cardiac dysfunction are common. These patients may initially require aggressive resuscitation to avoid hypovolemia and hypotension, but are also especially vulnerable to fluid overload.

A recent study compared functional outcomes in 160 patients after receiving either early goal-directed therapy guided by preload volume and cardiac output or monitoring with standard therapy [2^{••}]. In SAH patients with a poor clinical grade or coexisting cardiopulmonary complications, early goal-directed therapy reduced the likelihood of developing DCI and improved clinical outcomes. For patients with a good clinical grade, there was no benefit demonstrated to early goal-directed therapy over standard, less-invasive hemodynamic therapy.

Many recent studies have aimed at identifying optimal hemodynamic monitors and parameters that may be useful for guiding volume therapy. One study demonstrated a strong correlation between brain tissue oxygen response and cardiac response to fluid resuscitation

in patients with SAH [3]. The authors suggested that cardiac output and stroke volume variation (SVV) be incorporated in the hemodynamic monitoring of patients after SAH. They further asserted that the decision to administer a fluid challenge should reflect the baseline SVV and brain tissue oxygen pressure response to the fluid challenge. Another study demonstrated that mean global end-diastolic volume index (normal range 680–800 ml/m²) was an independent risk factor for the development of DCI (when less than 820 ml/m²) and pulmonary edema (when greater than 921 ml/m²) [4^{••}]. The authors concluded that global end-diastolic volume index should be maintained slightly above normal during fluid management of patients with SAH.

Interestingly, recent evidence suggests that the method of SAH treatment (surgical clipping or endovascular coiling) may greatly impact the patient's postoperative hemodynamic profile. Postoperative volume status and hemodynamics were compared in 73 patients treated with either surgical clipping or coiling, and patients who underwent clipping had a higher cardiac output and hypovolemia in the early postoperative period and a poorer preload responsiveness to volume [5[•]]. As such, after surgical clipping, patients required more intravenous volume to maintain normovolemia. These findings, which the authors attribute to increased surgical invasiveness and postoperative stress related to surgical clipping, have important implications for volume management in patients after SAH. Compared with endovascular coiling, patients after clipping may benefit from invasive hemodynamic monitoring to help guide volume resuscitation and achieve euvolemia. This is especially important during the period when patients are at risk for vasospasm and DCI.

IMPROVING NEUROLOGICAL OUTCOMES AFTER SUBARACHNOID HEMORRHAGE

The prevalence of cerebral vasospasm in patients with SAH ranges from 30 to 70%, and permanent morbidity or mortality results in up to 30% of these patients [6]. Although cerebral hypoperfusion may occur in the absence of cerebral vasospasm, cerebral vasospasm is an important cause of critically reduced cerebral perfusion [7]. Prevention of cerebral vasospasm is an important cornerstone of management after SAH. The risk of developing DCI is highest between days 4 and 10, and calcium channel antagonists are administered to decrease the risk.

A great deal of recent literature has aimed at determining etiologic factors that predict outcomes in patients after SAH. One study demonstrated that neuropeptides Argvasopressin and oxytocin are lower in patients with a poor outcome after SAH, possibly reflecting hypothalamic damage after SAH [8]. Furthermore, plasma levels of visfatin, an adipokine linked with inflammation, have been shown to predict the severity of SAH and have prognostic utility for clinical outcomes [9[•]].

Recent evidence has also suggested that cerebral vasospasm may be, in part, due to an acute endothelial dysfunction that results from an imbalance in the Arg and asymmetric dimethylarginine (ADMA) pathway [10[•]]. Interestingly, the ratio of plasma Arg:ADMA ratio has even been shown to predict mortality after SAH [11[•]]. Similarly, a recent study demonstrated that low levels of adiponectin from days 3 to 14 after SAH, which affects

nitric oxide production and endothelium-dependent vaso-relaxation, might be associated with the development of DCI [12].

Currently, there are efforts being made to develop techniques to clinical diagnostic tools to determine endothelial dysfunction. A few promising modalities include ultrasonographic imaging of endothelial-dependent flow-mediated dilation of the brachial artery and peripheral arterial tonometry [13]. Other models that attempt to predict cerebral vasospasm include various grading scales, and more recently, the application of a neural network model that will be examined in future studies [14]. One recent study identified younger age and early onset of vasospasm on transcranial Doppler as important predictors of severe vasospasm, and the authors recommend early and aggressive therapy in these patients [15].

It is unclear whether the choice of treatment modality for a ruptured aneurysm (surgical clipping or endovascular treatment) impacts the risk of developing vasospasm, although one recent retrospective study demonstrated that surgical clipping may be associated with an increased risk of vasospasm and delayed radiographic infarction [16^{*}]. These findings have generated some discussion and debate in the literature, and future prospective studies will be needed to confirm or rule out this assertion [17,18].

A great deal of research has focused on treatments that may reduce secondary brain injury and DCI after SAH in an effort to improve neurological outcomes and reduce mortality. Nimodipine is the only prophylactic drug that is known to reduce the risk of cerebral ischemia and improve neurological outcomes after SAH. The evidence for other calcium-blocking agents is inconclusive. Many recent studies attempt to identify other agents that may reduce the risk of cerebral vasospasm and improve neurological outcomes after SAH. Selective phosphodiesterase 3 inhibitors, such as cilostazol and milrinone, offer promise in the prevention of cerebral vasospasm after SAH because of their direct vasodilation and anti-inflammatory effects. Results of a recent meta-analysis demonstrated that cilostazol is effective in decreasing the incidence of symptomatic cerebral vasospasm, severe cerebral vasospasm, and cerebral vasospasm-related new cerebral infarctions after SAH [19^{**}]. Similarly, continuous infusion of milrinone significantly improved global cerebral oxygenation, and reduced the incidence of cerebral vasospasm during the critical 4 to 11 day postoperative period following cerebral aneurysm clipping surgery [20].

Magnesium is known to result in cerebral arterial dilation, and is a noncompetitive antagonist of calcium channels. Magnesium has therefore been suggested to be a potentially useful adjuvant to decrease the risk of vasospasm and improve neurological outcomes. A large randomized controlled trial however failed to demonstrate an improvement in clinical outcome with administration of magnesium after SAH, and the authors therefore recommended against the routine administration of magnesium [21]. Following this study, a meta-analysis demonstrated a decreased incidence of DCI in patients treated with magnesium after SAH, but failed to demonstrate any benefit with regards to neurological outcome, risk of cerebral vasospasm, or mortality [22]. The authors of the meta-analysis also advised against the routine use of magnesium in SAH. Other authors have pointed out, however, that every trial that failed to demonstrate an improvement in neurological outcomes or mortality did not compare magnesium-treated patients to a true placebo group

[23]. Nimodipine, a calcium channel blocker, was administered to patients in both the treatment and nontreatment groups. Therefore, it should not be surprising that a combination of two calcium channel antagonists failed to confer any additional benefit over nimodipine alone. The authors concluded that nimodipine might simply be the wrong partner for combination therapy with magnesium. Although current evidence suggests that nimodipine is the standard of care in SAH, a definitive clinical trial of nimodipine that examines neurological outcomes and mortality is needed [24].

The use of therapeutic hypothermia in the treatment of SAH has also been debated in the literature. Despite evidence from animal studies that therapeutic hypothermia may reduce secondary brain injury and the risk of cerebral vasospasm, hypothermia during aneurysm surgery has failed to demonstrate a clinical benefit [25]. One recent study examined Doppler middle cerebral artery (MCA) blood flow after induction of therapeutic hypothermia (33°, on average 5 days after SAH) in patients with increased intracranial pressure or DCI [25]. The authors demonstrated that therapeutic hypothermia resulted in decreased MCA blood flow, suggesting that therapeutic hypothermia may be useful for select patients. At present, more clinical data are needed before routine use of therapeutic hypothermia can be advocated.

Currently, the primary treatment of vasospasm is local injection of antispasmodic drugs. One study described their experience with 116 patients who underwent endovascular management for cerebral vasospasm [6]. The authors demonstrated that endovascular therapy is both well tolerated and effective in treating vasospasm and improving neurological outcomes. Both balloon angioplasty and nicardipine were shown to be equally effective, although nicardipine was less durable. Poor Hunt and Hess grades, preprocedure hypodensities, posterior circulation aneurysms, and the absence of neurological improvements after therapy were independent predictors of poor outcomes.

A recent review examined several clinical trials in which cerebral vasospasm was treated with intrathecal drug administration [26••]. The trials demonstrated that intrathecal administration of vasodilators or fibrinolytics was successful in lysing subarachnoid clots, attenuating cerebral vasospasm, and improving clinical outcomes. Furthermore, compared with systemic drug administration, intrathecal drug administration was associated with the delivery of higher concentrations of drug to the vessels in cerebral vasospasm with minimal systemic side-effects. Although intrathecal drug administration has not yet been widely adopted to treat cerebral vasospasm, it appears to be a promising alternative approach in the treatment of cerebral vasospasm.

There is evidence that some ICU interventions, such as early ambulation after SAH, improve clinical outcomes in elderly patients [27]. Other interventions used in patients after SAH, such as the intravenous administration of albumin, are commonly used among practitioners despite a lack of clinical trials that confirm its efficacy or safety [28]. Lastly, early preclinical data demonstrate that estrogen therapy can promote vasodilation by activating endothelial nitric oxide synthase, and may be neuroprotective after SAH [29]. However, at this time there is no clinical evidence supporting its routine use.

PULMONARY AND CARDIAC COMPLICATIONS

The clinical course for patients admitted to the ICU after SAH are often complicated by multisystem organ dysfunction, which may significantly worsen outcomes. A great deal of recent literature has focused on identifying and treating patients at risk for developing non-neurological complications.

Pulmonary complications are the most common non-neurological, medical cause of comorbidity after SAH. The incidence of acute respiratory distress syndrome (ARDS) in SAH may be as high as 37.6%, with a mortality rate in these patients exceeding 60% [30]. Neurogenic pulmonary edema is a life-threatening cause of ARDS, and should be considered in patients who develop ARDS after SAH [31]. Another recent study identified that more than 25% of patients after SAH develop postoperative aspiration pneumonia, which was associated with a significant (9.7%) risk of mortality [32].

Cardiac injury and left ventricular dysfunction are also common after SAH. The initiation of hypertension and hypervolemia to treat acute cerebral vasospasm may be especially problematic in these patients, and may result in significant morbidity. A recent prospective, multicenter cohort study determined that wall motion abnormalities on echo-cardiogram were independent risk factors for a poor outcome after SAH, explained in part by a higher risk of DCI [33]. The results of that trial mirrored prior retrospective studies, in which neurogenic stress cardiomyopathy after SAH was associated with a higher mortality and worsened functional outcomes [34]. Although it is unclear which patients are at increased risk for developing cardiogenic shock after SAH, a recent study [35**] demonstrated that prehospital use of β -blockers might decrease the risk.

There is new evidence that in some patients, namely young women with poor grade SAH, intra-aortic balloon pump placement may be a useful adjunct to ameliorate severe cardiogenic shock [36]. Future studies will need to better define the exact indication for this intervention, to confirm its efficacy, and to better define which patients might benefit from it. A ventricular assist device was also recently utilized to successfully facilitate myocardial and neurological recovery after SAH-related cardiogenic shock [37]. Other methods that may increase CBF, such as the NeuroFlo device, also show great promise but need to be studied further [38].

CONCLUSION

In recent years, there have been great advances in the postoperative ICU management of patients after SAH. Recent clinical studies have greatly focused on identifying prognostic indicators of poor outcomes and on establishing optimal hemodynamic monitors and parameters to guide volume therapy. Importantly, there are promising new treatment modalities aimed at improving neurological outcomes and treating non-neurological complications of SAH. As we improve our understanding of the pathophysiology and management of SAH and its clinical sequela, the next decade will hopefully see significant improvements in outcomes.

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KEY POINTS

- After SAH, brain tissue oxygen response and cardiac response to fluid resuscitation, as well as global end-diastolic volume index, may be useful for guiding volume therapy.
- Low plasma levels of Argvasopressin and oxytocin, and elevated levels of visfatin may have prognostic utility for predicting poor clinical outcomes after SAH.
- Recent evidence suggests that phosphodiesterase 3 inhibitors cilostazol and milrinone, magnesium therapy, and therapeutic hypothermia may be useful adjuncts in the treatment of cerebral vasospasm.
- Intrathecal administration of vasodilators and fibrinolytics has been successful in lysing subarachnoid clots, attenuating cerebral vasospasm, and improving clinical outcomes with minimal side-effects.
- Pulmonary disease and cardiogenic shock are common after SAH, are difficult to treat, and are significant risk factors for mortality.