SVOA Neurology

e-ISSN 2753-9180

ScienceVolks Open Access

Research Article

Proposal of an Institutional Protocol for the Management of Aneurysmal Subarachnoid Hemorrhage in a Tertiary Neurocritical Care Center

Mariana Rodrigues Martino, MD[1*]; Michel Franco Figueiredo, MD[1]; Bárbara Cozaro Valentim, MD[1]; Lucas Thodoro Kruze, MD[1]; Pedro Vicente Mesquita de Souza, MD[1]; Maria Eugênia Babbini Valentino Rosa, MD[1]; Fernando de Melo Filho, MD[1]

[1]Neurosurgery resident, Department of Neurosurgery, Hospital PUC Campinas, São Paulo, Brazil.

[2]Assistant Professor of Neurosurgery, Hospital PUC Campinas, São Paulo, Brazil.

***Corresponding Author:** Mariana Rodrigues Martino, Department of Neurosurgery, Hospital PUC Campinas, São Paulo, Brazil.

DOI: https://doi.org/10.58624/SVOANE.2026.07.010

Received: March 05, 2026

Published: March 25, 2026

Citation: Martino MR, Figueiredo MF, Valentim BC, Kruze LT, Souza PVM, Rosa MEBV, Filho FM. Proposal of an Institutional Protocol for the Management of Aneurysmal Subarachnoid Hemorrhage in a Tertiary Neurocritical Care Center. *SVOA Neurology* 2026, 7:2, 59-66. doi. 10.58624/SVOANE.2026.07.010

Abstract

Background: Aneurysmal subarachnoid hemorrhage (aSAH) is a severe neurological emergency characterized by high early mortality and substantial long-term disability. Beyond the initial hemorrhage, secondary mechanisms including delayed cerebral ischemia (DCI), vasospasm, hydrocephalus, and systemic complications significantly influence prognosis.

Objective: To propose a structured institutional protocol for the management of aSAH based on contemporary high-impact literature and international guideline recommendations.

Methods: A structured narrative review was conducted using PubMed and Scopus databases covering publications from 2020 to 2025. In addition, foundational references directly informing endovascular management principles were included regardless of publication year. Search terms included “aneurysmal subarachnoid hemorrhage,” “vasospasm,” “delayed cerebral ischemia,” “lumbar drainage,” “nimodipine,” and “neurocritical care protocol.” Inclusion criteria comprised international guidelines, randomized clinical trials, systematic reviews, and high-impact observational studies. Case reports, pediatric-only studies, and animal studies were excluded. Foundational guideline documents and landmark trials directly informing protocol construction were incorporated. Evidence was synthesized into a phase-based multidisciplinary institutional care pathway.

Results: Evidence synthesis supported inclusion of early imaging confirmation, structured severity grading, strict pre-occlusion blood pressure control, universal nimodipine therapy, selective seizure prophylaxis, systematic transcranial Doppler surveillance, individualized early lumbar drainage, criteria-based external ventricular drainage, metabolic optimization, and tiered escalation strategies including induced hypertension, endovascular intervention, and intravenous milrinone in refractory vasospasm.

Conclusion: Institutional standardization grounded in contemporary evidence may reduce variability in care delivery and promote consistent neurocritical management of patients with aSAH.

Keywords: Aneurysmal Subarachnoid Hemorrhage; Delayed Cerebral Ischemia; Vasospasm; Lumbar Drainage; Neurocritical Care; Protocol Proposal.

Introduction

Aneurysmal subarachnoid hemorrhage (aSAH) represents a catastrophic cerebrovascular event with significant morbidity and mortality worldwide. Despite advances in microsurgical and endovascular aneurysm treatment, outcomes remain heavily influenced by early brain injury and secondary complications such as rebleeding, acute hydrocephalus, cerebral vasospasm, and delayed cerebral ischemia (DCI) (1). DCI, in particular, remains a principal determinant of long-term functional outcome.

The 2023 American Heart Association/American Stroke Association (AHA/ASA) guideline emphasizes rapid diagnosis, prompt aneurysm occlusion, intensive neurological monitoring, and prevention of secondary ischemic injury as central pillars of management (1). Prognostic stratification tools including the modified Fisher scale and the modified Hunt–Hess grading scale provide structured risk assessment and correlate with complications such as hydrocephalus and vasospasm (2,3).

Emerging high-impact literature has further explored adjunctive strategies, including lumbar cerebrospinal fluid (CSF) drainage for reduction of secondary infarction (9–11) and pharmacologic escalation with intravenous milrinone for refractory vasospasm (5– 8). However, translation of these elements into a cohesive operational institutional framework remains heterogeneous.

Given this context, we present a proposal for a structured institutional protocol for aSAH management integrating guideline-based recommendations and contemporary high-impact evidence into a multidisciplinary neurocritical care pathway.

Methods

A structured narrative review was conducted using PubMed and Scopus databases, by single reviewer with supervision. The search period included January 2020 through 2025. Search terms were: “aneurysmal subarachnoid hemorrhage,” “vasospasm,” “delayed cerebral ischemia,” “lumbar drainage,” “nimodipine,” and “neurocritical care protocol.”

Priority inclusion criteria were recent international clinical guidelines, particularly the 2023 American Heart Association/American Stroke Association guideline (1), randomized controlled trials, systematic reviews, meta-analyses, and high-impact cohort studies relevant to adult aSAH management, including pharmacologic escalation strategies for vasospasm and delayed cerebral ischemia (5–8). Exclusion criteria included case reports, animal studies, pediatric-only studies, and publications not addressing clinical management strategies

Foundational documents directly informing protocol construction, including the 2023 AHA/ASA guideline (1) and key trials on lumbar drainage and vasospasm management (4–11), were incorporated. Evidence was synthesized into a phase-oriented institutional operational protocol.

Results

The 2023 American Heart Association/American Stroke Association (AHA/ASA) guideline emphasizes prompt diagnosis, early aneurysm treatment, neurocritical monitoring, and prevention/management of secondary complications such as DCI and vasospasm. (1)

The guideline further recommends early aneurysm occlusion to reduce rebleeding risk, management in specialized neurocritical care settings, universal administration of oral nimodipine for 21 days, treatment of symptomatic vasospasm with hemodynamic augmentation, and structured surveillance for delayed cerebral ischemia (1).

Regarding prognostic stratification, Oliveira et al. (2011) evaluated the revised/modified Fisher approach in aneurysmal SAH and reported its utility for prognostic assessment in relation to vasospasm risk during the acute course, using serial clinical examinations and transcranial Doppler monitoring in their prospective design. (2) Al-Mufti et al. (2024) proposed the modified Hunt–Hess grading scale and reported improved prognostic discrimination compared with the traditional scale, particularly in patients with severe aSAH, based on retrospective outcome analysis (3).

Pierot et al. (2010) reviewed advances in endovascular management of vasospasm, describing balloon angioplasty and intra-arterial vasodilator therapy as established options. As Fraticelli et al. (2008) reported clinical improvement and angiographic reversal of vasospasm following intravenous milrinone administration (5). Santos-Teles et al. (2020), in a systematic review, concluded that available evidence indicates possible efficacy of milrinone for vasospasm treatment (6). Additionally, Lakhal et al. (2021) (MILRISPASM study) evaluated intravenous milrinone combined with induced hypertension in vasospasm management (7). And as Baang et al. (2025), which analyzed early intravenous milrinone as first-line therapy for cerebral vasospasm or delayed cerebral ischemia, reported clinical improvement (8).

Regarding Lumbar Cerebrospinal Fluid Drainage, Al-Tamimi et al. (2012), in the LUMAS randomized controlled trial, reported that lumbar CSF drainage reduced delayed ischemic neurological deficit and improved early clinical outcome, although no statistically significant improvement in 6-month functional outcome was observed (10). Further, Hulou et al. (2022), in a systematic review and meta-analysis including 17 studies, reported that lumbar drainage was associated with improved short-term functional outcome, reduced mortality, lower incidence of clinical vasospasm, and decreased delayed ischemic neurological deficits compared with no drainage (11).Wolf et al. (2023), in the EARLYDRAIN randomized clinical trial, also demonstrated that early lumbar drainage initiated within 72 hours was associated with a lower rate of unfavorable neurological outcome (9).

Discussion

The Institutional Protocol Emergency Evaluation and Diagnostic Strategy

Patients presenting with acute headache and clinical suspicion for subarachnoid hemorrhage undergo immediate non-contrast head computed tomography (CT), within 20 minutes of emergency department arrival (1). When SAH is confirmed, CT angiography or digital subtraction angiography is performed to identify the aneurysmal source.

CT sensitivity is highest within the first six hours after symptom onset and declines between six and twenty-four hours, with further reduction beyond twenty-four hours. In cases where clinical suspicion persists despite a negative CT, particularly beyond six hours, lumbar puncture is recommended for diagnostic clarification and becomes strongly indicated after twenty-four hours after symptom onset. (1)

Clinical Severity Grading and Monitoring

Upon admission, patients are graded using the Hunt–Hess scale, the World Federation of Neurosurgical Societies (WFNS) scale, and the modified Fisher scale. Serial reassessment is important. The modified Fisher scale correlates with vasospasm and DCI risk (2), and the modified Hunt–Hess grading improves prognostic stratification (3). Continuous neurological surveillance, including Glasgow Coma Scale assessment, pupillary examination, focal motor evaluation, and language assessment when applicable, is performed hourly in the pre-occlusion phase and adjusted according to clinical stability.

Mandatory laboratory and bedside investigations must be obtained immediately upon hospital arrival to allow comprehensive systemic assessment and early identification of reversible complications that may influence neurological and systemic outcomes. The initial diagnostic panel should include a complete blood count; serum electrolytes, particularly sodium and potassium; renal function tests (urea and creatinine); blood glucose levels; and a coagulation profile including prothrombin time/international normalized ratio (PT/INR) and activated partial thromboplastin time (aPTT). In critically ill or intubated patients, arterial blood gas analysis should be performed to assess oxygenation, ventilation, and metabolic status. A chest radiograph and a 12-lead electrocardiogram (ECG) are also required at admission as part of the comprehensive initial evaluation recommended in contemporary guideline-based care (1).

Electrocardiographic evaluation is mandatory in all patients to detect neurogenic cardiac abnormalities, including arrhythmias and repolarization changes, and to establish a baseline for continuous cardiac monitoring, as cardiac dysfunction is a recognized systemic complication of aneurysmal subarachnoid hemorrhage (1). Serum troponin levels should be obtained in critically ill patients, in those presenting with ECG abnormalities, or in cases of hemodynamic instability, given the documented occurrence of neurogenic myocardial injury in this population (1). Chest radiography provides essential baseline information for intensive care planning and assists in the identification of aspiration events or neurogenic pulmonary edema, which are described extracranial complications in the acute phase of aSAH (1).

Prevention of systemic complications

Mechanical prophylaxis for venous thromboembolism (VTE), including graduated compression stockings or intermittent pneumatic compression devices, should be initiated immediately upon admission. Pharmacologic prophylaxis with prophylactic- dose unfractionated heparin or low-molecular-weight heparin may be introduced only after definitive aneurysm exclusion (1).

Stress ulcer prophylaxis is indicated in critically ill patients, particularly those receiving mechanical ventilation, presenting with coagulopathy, or undergoing corticosteroid or anticoagulant therapy, and may be achieved with proton pump inhibitors or H2 receptor antagonists according to institutional practice (1).

Prevention of pressure injuries and immobility-related complications requires scheduled repositioning at least every two hours, use of skin-protection devices, early nutritional assessment, and mobilization as clinically feasible (1).

Serial laboratory surveillance is mandatory and includes daily or 12–24-hour serum sodium monitoring, hemogram and renal function assessment according to clinical evolution, and capillary glucose measurement per institutional protocol. Electrolyte disturbances, particularly dysnatremias, are common after aSAH and are associated with increased risk of neurological complications; therefore, correction must be gradual to avoid osmotic shifts. Coagulopathies increase the risk of rebleeding, and accurate renal function assessment is essential for contrast-based imaging and endovascular therapeutic planning (1).

Hemodynamic Stabilization and Rebleeding Prevention

Initial measures include clinical stabilization, evaluation for advanced life support, hemodynamic stabilization, and airway management when indicated. Hypoxemia is treated aggressively due to its association with worse neurological outcomes. Patients should be managed in an intensive care setting, preferably a neurocritical care unit. Bed rest, adequate analgesia, management of psychomotor agitation when present, and invasive blood pressure monitoring are required.

Before aneurysm exclusion, strict blood pressure control is implemented to reduce rebleeding risk. Although no universal fixed threshold exists, systolic blood pressure values exceeding 160–180 mmHg have been associated with increased rebleeding risk. Operational targets include maintaining systolic blood pressure between 120 and 160 mmHg, with continuous neurological surveillance, avoiding mean arterial pressure below 65 mmHg, and preventing abrupt reductions (1).

Continuous neurological surveillance must be maintained throughout both the pre- and post-aneurysm exclusion phases. In the hyperacute period, any neurological change should be interpreted as a potentially severe event, frequently reflecting acute hydrocephalus, rebleeding, intracranial hypertension, or early delayed cerebral ischemia, and therefore requires immediate neurosurgical reassessment (1).

During the pre-occlusion phase, hourly documentation of neurological status is recommended, including Glasgow Coma Scale (GCS), Hunt–Hess grading, World Federation of Neurosurgical Societies (WFNS) scale, pupillary examination, focal motor assessment, and language evaluation when applicable (1–3). The modified Fisher and modified Hunt–Hess scales contribute to prognostic stratification and risk assessment for complications such as vasospasm and hydrocephalus, reinforcing the importance of serial grading (2,3). Once the aneurysm has been excluded and the patient is clinically stable, examinations may be performed every two hours; however, surveillance must return to hourly assessments in cases of high clinical risk, hemodynamic instability, the defined risk window for delayed cerebral ischemia, or any fluctuation in level of consciousness, new focal neurological deficit, or unexplained deterioration (1). Immediate neurological evaluation is mandatory whenever acute worsening is detected.

Seizure Prophylaxis

Routine prophylactic antiseizure medication is not recommended in all patients (1). Therapy is reserved for documented clinical or electrographic seizures, intracerebral hematoma with cortical involvement or significant mass effect, or extensive cortical SAH. Levetiracetam is preferred due to its pharmacological profile, administered at 500–1000 mg every 12 hours orally or intravenously, adjusted for renal function.

Treatment duration is individualized, typically limited to the acute phase (seven days) in the absence of recurrent seizures. (1)

Nimodipine Therapy

All patients receive nimodipine 60 mg every four hours for twenty-one days unless contraindicated. Nimodipine reduces clinically relevant ischemic events and improves functional outcomes despite not preventing angiographic vasospasm (1).

Immediate neurosurgical assessment

All confirmed SAH patients require early neurosurgical evaluation. Delays in aneurysm occlusion are associated with worse functional prognosis (protocol rationale; guideline framework).(1)

Aneurysm Exclusion

Early aneurysm occlusion by clipping or endovascular therapy is recommended, ideally within 24–72 hours, to reduce rebleeding and permit safer management of complications (1). Treatment modality selection considers aneurysm anatomy, associated intraparenchymal hematoma, suspicion of vasospasm, and patient clinical status.

Lumbar Drainage

Lumbar CSF drainage is incorporated as an individualized adjunct following aneurysm exclusion. Drainage is preferably initiated within 48 hours after aneurysm treatment and no later than 72 hours from symptom onset. Continuous drainage is maintained at approximately 5 mL per hour (≈120 mL per day) for 5–7 days, extendable up to 10 days depending on CSF clearance and clinical course (9–11).

Hydrocephalus and External Ventricular Drainage (EVD)

External ventricular drainage (EVD) is indicated in patients with aneurysmal subarachnoid hemorrhage who develop clinical and radiological evidence consistent with acute hydrocephalus. Clinical criteria include a decrease in level of consciousness defined as a ≥2-point reduction in the Glasgow Coma Scale, acute neurological deterioration not attributable to sedation, progressive somnolence or coma, and signs of intracranial hypertension such as refractory headache, projectile vomiting, or pupillary abnormalities (1). Radiological criteria, interpreted in conjunction with clinical findings, include ventricular enlargement compatible with acute hydrocephalus, sulcal effacement, increased Evans index, and hydrocephalus associated with high modified Fisher grades and/or intraventricular hemorrhage; acute hydrocephalus in patients with severe SAH (WFNS IV–V), even when neurological examination is limited, also supports intervention (1,2). When these criteria are met, EVD placement should not be delayed.

Safe EVD management requires strict nursing protocols, including accurate leveling of the drainage system at the foramen of Monro according to medical prescription, maintenance of a closed and sterile system, avoidance of disconnections, verification of catheter patency, and hourly documentation of cerebrospinal fluid output, appearance, and neurological status (1). Infection prevention measures include rigorous hand hygiene, use of clean, dry, occlusive dressings—preferably chlorhexidine-impregnated when available—daily sterile dressing changes with aqueous chlorhexidine if such dressings are unavailable, inspection of the insertion site for inflammatory signs, and sterile disposal of drainage bag contents with system isolation. During patient mobilization, the EVD must be temporarily closed after emptying and documenting reservoir volume to prevent overdrainage and filter contamination, and catheter traction must be avoided. Immediate communication with the neurosurgical team is mandatory in cases of absent drainage for more than two hours, excessive drainage, neurological worsening, fever, or suspected infection (1).

Weaning should be attempted after clinical stabilization; if EVD occlusion is tolerated for 48 hours without neurological deterioration and follow-up computed tomography at 24–48 hours shows no recurrent hydrocephalus, the device may be removed. Failure of weaning warrants cerebrospinal fluid analysis and planning for definitive cerebrospinal fluid diversion, preferably ventriculoperitoneal shunting, provided at least two cerebrospinal fluid samples show no evidence of infection and two cultures are negative prior to internalization (1).

Vasospasm and Delayed Cerebral Ischemia

Vasospasm typically occurs between days 3 and 21, peaking around days 7–10 (1). Serial transcranial Doppler monitoring is performed daily from day 3 to day 14 and Increase to every 12 hours in high-risk patients or when vasospasm is detected. Digital subtraction angiography is considered in refractory cases or candidates for endovascular therapy.(4)

Patients at increased risk for the development of cerebral vasospasm and delayed cerebral ischemia (DCI) include those presenting with thick subarachnoid hemorrhage as classified by a high modified Fisher grade, associated intraventricular hemorrhage, severe clinical presentation defined as WFNS grades IV–V, younger age, and delayed aneurysm exclusion (1–3). These variables are consistently associated with greater vasospasm burden and worse neurological outcomes, reinforcing the need for intensified surveillance in this subgroup (1,2).

Transcranial Doppler (TCD) ultrasonography constitutes a central noninvasive tool for serial surveillance. Interpretation is based not only on absolute velocities but also on dynamic trends and index calculations (1). In the middle cerebral artery (MCA), mean flow velocities <120 cm/s with a Lindegaard index <3 are considered absent vasospasm; velocities between 120–149 cm/s with Lindegaard index 3–4 indicate mild vasospasm; 150–199 cm/s with index 4–6 indicate moderate vasospasm; and ≥200 cm/s with Lindegaard index ≥6 indicate severe vasospasm. The Lindegaard index, calculated as the ratio between MCA mean velocity and extracranial internal carotid artery velocity, distinguishes true vasospasm from hyperemia. For the anterior cerebral artery, mean velocities ≥120 cm/s suggest vasospasm, although interpretation may be limited by insonation angle and anatomy.

In the posterior circulation, vasospasm is suggested by posterior cerebral artery mean velocities ≥100 cm/s or progressive increases, while basilar artery velocities ≥70 cm/s with a Soustiel index ≥2 indicate vasospasm; the Soustiel index represents the ratio between basilar and extracranial vertebral artery velocities (1). Dynamic criteria strengthen diagnostic suspicion, particularly when there is an increase ≥50 cm/s within 24 hours, ≥30 cm/s within 48 hours, significant hemispheric asymmetry, or concordance with clinical deterioration (1). In practical terms, an MCA velocity ≥150 cm/s and a Lindegaard index ≥3 are considered indicative of clinically significant vasospasm, and ascending velocity trends are more informative than isolated values (1).

Systemic and metabolic optimization is fundamental in all patients at risk for DCI. Strict euvolemia must be maintained, avoiding both hypovolemia—which compromises cerebral perfusion—and prophylactic hyperhydration, which may increase the risk of pulmonary and cerebral complications (1). Fluid balance should be closely monitored, with particular attention to high urine output in patients who may subsequently receive vasodilatory agents such as milrinone. Electrolyte disturbances must be promptly corrected, maintaining normonatremia, serum potassium levels ≥4.0 mmol/L, and magnesium levels ≥0.8 mmol/L, as electrolyte instability may exacerbate arrhythmias and impair cerebral perfusion (1).

Following confirmation of vasospasm, structured severity stratification should consider angiographic burden (moderate versus severe), presence of neurological deficit, focal versus diffuse distribution, and systemic comorbidities including cardiac, renal, and infectious conditions. This stratification guides intensification of monitoring and early multidisciplinary discussion with interventional neuroradiology (1,4). In patients with symptomatic vasospasm after aneurysm exclusion, induced hypertension is indicated and should be titrated to neurological response; although specific mean arterial pressure targets are not universally standardized, some institutional practices maintain values around 90–100 mmHg under close monitoring (1).

In selected cases of symptomatic vasospasm or DCI unresponsive to initial hemodynamic optimization, intravenous milrinone may be administered as part of an escalation protocol supported by contemporary literature (5–8). The Montreal protocol consists of an initial intravenous bolus of 0.1 mg/kg administered over 10 minutes, followed by continuous infusion beginning at 0.75 µg/kg/min. Dose escalation may proceed stepwise up to 1.25 µg/kg/min, with reported maximum infusion rates of 2.0 µg/kg/min depending on neurological response and systemic tolerance. Continuous cardiac monitoring is mandatory, and vasopressor support is frequently required due to the vasodilatory effects of milrinone (5–8).

Conclusion

This proposal integrates the 2023 AHA/ASA guideline framework (1) with contemporary evidence addressing prognostic grading (2,3), vasospasm surveillance and intervention (4), lumbar drainage (9–11), and intravenous milrinone in refractory DCI (5–8). By synthesizing these elements into a structured operational pathway, the protocol aims to reduce variability in institutional management and enhance multidisciplinary coordination.

This institutional protocol provides a detailed and standardized pathway for the management of aneurysmal subarachnoid hemorrhage in a tertiary-care setting. It defines actionable steps from emergency diagnosis through neurocritical stabilization, aneurysm management, structured monitoring, prophylaxis bundles, and complication- directed escalation strategies. By reducing variability and formalizing multidisciplinary workflows, the protocol aims to support timely intervention and consistent delivery of care aligned with the referenced literature.

Funding

None.

Competing Interests

The authors declare no competing interests.

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