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Literature Review

Diagnosis and Management of Cerebral Vasospasm Following Aneurysmal SAH

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Abstract

Cerebral Vasospasm, characterized by the progressive constriction of cerebral arteries, often occurs following a subarachnoid hemorrhage (SAH) and is a leading cause of morbidity and mortality in affected patients. This condition can be resulted in cerebral ischemia, the severity of which correlates with the degree of vasospasm. The underlying pathophysiology involves the encasement of arteries by blood clots, although the intricate interactions between the hematoma and adjacent structures remain incompletely understood. The delayed onset of vasospasm offers a potential window for preventive interventions. However, recent randomized controlled trials have been discouraging, as they failed to demonstrate any significant improvement in patient outcomes with the use of clazosentan (an endothelin antagonist), simvastatin (a cholesterol-lowering agent), or magnesium sulfate (a vasodilator). Current best practices for managing vasospasm include minimizing ischemia by maintaining adequate blood volume and pressure, administering nimodipine (a calcium channel blocker), and, when necessary, performing balloon angioplasty. Over the past two decades, advancements in the management of vasospasm have significantly reduced associated morbidity and mortality rates. Nevertheless, vasospasm remains a critical determinant of clinical outcomes following aneurysmal rupture.

Keywords: Diagnosis, cerebral vasospasm, management

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Introduction

Cerebral vasospasm represents a severe complication following subarachnoid hemorrhage (SAH), often leading to delayed cerebral ischemia and infarction, and continues to be a central concern in neurocritical care.[1] The diagnosis of cerebral vasospasm poses considerable challenges due to its intricate pathophysiology and the difficulties associated with its early identification. Nevertheless, recent advancements in imaging modalities, such as CT perfusion, MRI, and transcranial Doppler ultrasonography (TCD), have significantly enhanced the precision and speed of detection. These technological improvements have created new opportunities for timely interventions, thereby improving the potential for more effective clinical management of this critical condition. Vasospasm is observed in roughly 40–50% of patients following SAH, with its peak occurrence typically between days 4 and 5 after the initial bleeding event.[2] Despite extensive research, the precise mechanisms driving vasospasm are still being elucidated. Earlier investigations have established that vasospasm is a key contributor to delayed cerebral ischemia, one of the most dreaded outcomes associated with SAH. Vasospasm is essentially a reactive process marked by abnormal or localized constriction of cerebral arteries. This phenomenon is thought to be resulted from the release of hemolytic byproducts, which activate smooth muscle cells in the arterial walls. These effects are primarily mediated through calcium

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ion (Ca^{2+}) signaling pathways and the action of prostaglandins, particularly prostaglandin E_2 (PGE_2).[3]

Cerebral vasospasm is strongly influenced by various predisposing factors, with the volume of subarachnoid blood detected on CT imaging being a critical determinant. This highlights the necessity of timely and precise diagnostic imaging in the clinical management of high-risk patients. Contemporary therapeutic approaches emphasize both the prevention and treatment of vasospasm. Nimodipine is regarded as the cornerstone of prophylactic therapy, while hemodynamic augmentation through induced hypertension is commonly employed to manage symptomatic cases. Aneurysmal rupture leading to SAH affects approximately 10 in 10,000 individuals annually, with a mortality rate nearing 40%.[4] Survivors of the initial hemorrhagic episode face a substantial risk of neurological deficits due to delayed cerebral vasospasm. Approximately two-thirds of SAH patients develop arterial narrowing indicative of vasospasm between days 3 and 14 following the rupture; however, only about one-third of these individuals manifest clinically significant neurological symptoms.[5]

Patients with posterior circulation aneurysms are at heightened risk, with nearly 75% experiencing symptomatic vasospasm. The temporal profile of complications varies, with intracranial hemorrhage or vasospasm peaking within the first 24 to 48 hours after traumatic injury and rebleeding risk highest in the first 24 hours post-rupture.6 Additionally, patients undergoing craniotomy for tumor resection are vulnerable to vasospasm or hemorrhage within 48 to 72 hours post-surgery.[7] While existing therapies are effective, novel strategies—including endothelin receptor antagonists, magnesium sulfate, statins, and endovascular interventions like transluminal angioplasty—are being explored.[8–10] Despite progress, cerebral vasospasm remains a leading cause of morbidity and mortality in SAH patients, necessitating ongoing research and innovation in neurocritical care.[11,12]

Literature Review

Definition

Cerebral vasospasm is characterized by abnormal and sustained constriction of cerebral arteries, commonly occurring after SAH.[13] This narrowing of blood vessels significantly threatens cerebral perfusion, increasing the risk of ischemia, which can lead to stroke, neurological deficits, or death if not promptly managed. Symptomatic vasospasm presents as new neurological deficits or a decline in consciousness, both resulting from ischemic injury.[14] Accurate diagnosis requires ruling out other potential causes of deterioration, such as hydrocephalus, seizures, metabolic disturbances, infection, or excessive sedation. Angiographic vasospasm is defined by moderate to severe arterial narrowing seen on DSA, distinct from changes caused by atherosclerosis, catheter-induced spasm, or congenital vascular anomalies.[15] Typically associated with blood flow velocities exceeding 120 cm/s, it emerges between days 3 and 14 following hemorrhage and serves as a critical predictor of clinical outcomes and prognosis.

Risk factor

The extent of clot burden within the subarachnoid space is the most important predictor for the development of cerebral vasospasm following SAH. The Fisher scale and its modified version are commonly used to evaluate this risk by

Table 1. Stages of Cerebral Vasospasm Development[2,16]

Timeframe	Stage of development
Day 0	Initiation - Following SAH due to aneurysm rupture
Days 3-5	Early Vasospasm - Spasms begin to develop, possibly with mild symptoms
Days 6-8	Peak Vasospasm - Maximum intensity, highest risk of ischemic stroke.
Days 9-14	Resolution - Spasms subside, clinical symptoms decrease
After Day 14	Post-Vasospasm Recovery - Blood vessels return to normal, recovery from brain damage progresses
After 2 Weeks	Rehabilitation - Patients may require rehabilitation for residual effects

classifying CT findings; the modified Fisher scale assigns scores from 0 to 4 based on subarachnoid blood thickness and the presence of IVH, offering a more precise assessment of vasospasm likelihood. Additional risk factors include delayed clot resolution, which is challenging to assess clinically, along with variables such as loss of consciousness at aneurysm rupture, poor neurological status on admission, and comorbidities like smoking, diabetes mellitus, hyperglycemia, hypertension, younger age, and cocaine use. Aneurysm location also plays a significant role, with distal anterior cerebral artery aneurysms posing a higher risk due to their proximity to major cerebral vessels vulnerable to post-hemorrhagic hemodynamic changes. In a study of 370 SAH patients, left ventricular hypertrophy and hypertension were identified as strong predictors of severe vasospasm, underscoring the importance of proactive monitoring and management.[17] Understanding these risk factors is crucial for developing targeted strategies to prevent and treat cerebral vasospasm, ultimately reducing its incidence and improving clinical outcomes in SAH patients.[18]

Pathophysiology

Cerebral vasospasm can be classified by type and underlying cause. Symptomatic vasospasm involves clinical signs such as headache, neurological deficits, or altered consciousness resulting from arterial narrowing, which, if untreated, can progress to delayed cerebral ischemia and infarction.[19] Angiographic vasospasm, in contrast, is detected through imaging modalities like angiography and may not present with symptoms but can still impair cerebral blood flow and lead to ischemic injury.[19] The etiology is multifactorial, with SAH as the primary trigger due to blood accumulation in the subarachnoid space and the release of vasoactive substances like oxyhemoglobin, which induce prolonged vasoconstriction.[20] Trauma, surgical manipulation, and infection may also lead to vasospasm via mechanical injury, inflammation, or endothelial disruption.[20] Inflammatory mediators, cytokines, and oxidative stress further impair vascular tone and function, while hydrocephalus may elevate intracranial pressure, increasing vasospasm risk. Genetic predisposition and endothelial dysfunction also play contributory roles.

At the core of vasospasm pathophysiology is smooth muscle contraction, largely driven by increased intracellular calcium within vascular smooth muscle cells. Following SAH, blood breakdown products such as oxyhemoglobin and deoxyhemoglobin heighten contractility, resulting in sustained vasoconstriction.[19] Concurrent endothelial injury impairs the release of vasodilators like NO and prostacyclin while promoting vasoconstrictors such as endothelin-1, worsening arterial narrowing. Inflammation further exacerbates this process through cytokine-mediated smooth muscle activation and endothelial damage, increasing the likelihood of delayed cerebral ischemia.[19] Recurrent vasospasm may eventually lead to vascular remodeling, marked by structural alterations that impair vessel reactivity. Additionally, early brain injury (EBI), occurring shortly after hemorrhage or trauma, initiates oxidative stress and disrupts cerebral autoregulation, amplifying the severity and persistence of vasospasm. This complex interplay of vascular, cellular, and biochemical mechanisms underlies the development of cerebral vasospasm and informs strategies for its clinical management.

Clinical manifestation

Cerebral vasospasm presents clinically with symptoms resulting from reduced cerebral perfusion, commonly beginning with a sudden, severe "thunderclap" headache that may resemble the initial SAH episode. As the condition progresses, focal neurological deficits such as hemiparesis, aphasia, or sensory disturbances may appear, along with changes in consciousness ranging from confusion to coma. Seizures, either focal or generalized, can occur due to ischemic injury, while cognitive and behavioral changes—such as memory loss and irritability—may also be observed. In severe or prolonged cases, delayed cerebral ischemia may develop, leading to permanent deficits including motor, sensory, language, and cognitive impairments, with possible visual disturbances like blurred vision or

field loss. Prompt recognition and management of these signs are critical to reducing morbidity and improving outcomes.

Diagnosis

Cerebral vasospasm is a critical complication of aSAH that requires vigilant monitoring and timely intervention, guided by AHA/ASA recommendations for managing vasospasm and DCI. Standard surveillance includes TCD, with CTA, CT perfusion, EEG, and biochemical markers used for further assessment in complex cases. Management strategies center on enteral nimodipine and maintaining euvolemia, with more aggressive treatments like induced hypertension or endovascular therapy reserved for severe or refractory cases, while routine use of statins, magnesium, or prophylactic hemodynamic manipulation is not recommended.

The American College of Radiology (ACR) Appropriateness Criteria provide a framework for selecting optimal imaging strategies in patients with suspected cerebral vasospasm. Standard imaging methods typically encompass cervicocerebral angiography and contrast-enhanced CTA. Supplementary imaging modalities that may be considered appropriate include transcranial Doppler ultrasound, contrast-enhanced head MRI perfusion, non-contrast head MRI, and non-contrast head CT.[2] These diagnostic tools play a critical role in facilitating precise identification and effective management of cerebral vasospasm, as well as mitigating potential complications arising from this condition.

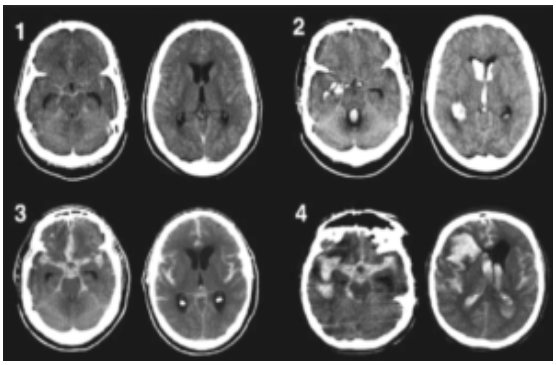

Figure 1. Modified Fisher CT Grading Scale

FISHER GRADE

Group	Blood on CT
1	No SAH detected
2	Diffuse or vertical layer of subarachnoid blood < 1mm thick
3	Localised clot and/or vertcal layer within the subarachnoid space > 1mm thick
4	ICH or IVH with diffuse or no SAH

MODIFIED FISHER GRADE

		No SAH	Thin SAH	Thick SAH	IVH
0	No SAH; no IVH	+	-	-	-
1	Thin diffuse or focal SAH, no IVH	-	+	-	-
2	Thin diffuse or local SAH with IVH	-	+	-	+
3	Thick focal or diffuse SAH, no IVH	-	-	+	-
4	Thick local or diffuse SAH with IVH	-	-	+	+

Figure 2. Differences Between the Classic and Modified Fisher Scales.
(Source: https://pbrainmd.wordpress.com/2015/02/)

The modified Fisher scale predicts the risk of vasospasm in aSAH patients by scoring the extent of SAH and presence of IVH, with higher scores indicating greater risk. CT perfusion imaging has shown superior accuracy in detecting delayed cerebral ischemia compared to non-contrast CT and CTA. However, postoperative intra-aortic counterpulsation balloon therapy has not demonstrated significant benefits over hypervolemic therapy in improving outcomes.

Transcranial Doppler ultrasonography (TCD) is the primary non-invasive method for detecting cerebral vasospasm, relying on increased blood flow velocity as arteries narrow. It is commonly used in SAH patients to identify early signs of vasospasm through velocity changes detected via Doppler frequency shifts. TCD employs a 2 MHz ultrasound probe applied to cranial acoustic windows, such as transtemporal or submandibular regions, where the skull is thinner. TCD is highly effective in detecting proximal cerebral vasospasm by identifying segmental and diffuse increases in blood flow velocity, particularly in vessels like the MCA, where velocities above 120 cm/s suggest vasospasm and those over 200 cm/s indicate severe vasospasm. Differentiating vasospasm from hyperemia requires comparing intracranial velocities with those of the cervical ICA, using parameters like the Lindegaard ratio and SVIRI ratio. While TCD is reliable for major arteries, it is less effective for distal branches, limiting its correlation with perfusion

Table 1. Breakdown of Diagnostic Modalities for Cerebral Vasospasm Cases

Modality	Description	Advantages	Limitations
Serial Psychological Assessment	Repeated evaluation of cognitive function and mental status	Non-invasive, identifies early neurocognitive changes	Subjective, dependent on patient cooperation, does not directly detect vasospasm
TCD (Transcranial Doppler Ultrasound)	A non-invasive ultrasound tech-nique that measures cerebral blood flow velocity.	Real-time monitoring, portable, cost-effective	Operator-dependent, indirect measurement, limited to large vessels
CTA (CT Angiography)	Imaging technique that provides detailed images of brain blood vessels	High spatial resolution, can detect vasospasm and infarction	Involves radiation and contrast dye, less suitable for continuous monitoring
CTP (CT Perfusion Imaging)	Imaging that evaluates cerebral blood flow, volume, and transit time	Can detect areas at risk of infarction, complements CTA	Involves radiation, contrast dye, limited availability
MRI (Magnetic Resonance Imaging)	A non-invasive imaging technique using a magnetic field to produce brain images	No radiation exposure, high tissue resolution	Time-consuming, expensive, does not directly detect vasospasm
PWI (Perfusion-Weighted Imaging)	Assesses brain perfusion using MRI to evaluate blood flow and volume	Non-invasive, no radiation, good tissue characterization	Expensive, longer scan time, limited availability
DSA (Digital Subtraction Angiography)	An invasive technique providing detailed images of blood vessels using contrast dye	Gold standard for vasospasm detection, high accuracy	Invasive, risk of complications, expensive, time-consuming
Brain Tissue Oxygenation Monitoring	Monitors brain tissue oxygenation using sensors placed in brain parenchyma	Direct tissue oxygenation monitoring, identifies hypoxia	Invasive, risk of infection, limited availability
Cerebral Microdialysis Catheter	Measures metabolites in brain interstitial fluid to detect ischemia	Sensitive to metabolic changes, local monitoring	Invasive, risk of infection, limited localized data
Near-Infrared Spectroscopy (NIRS)	A non-invasive method using near-infrared light to monitor cerebral oxygenation	Continuous monitoring, portable, non-invasive	Limited penetration depth, indirect measurement, sensitive to artifacts

deficits seen in CBF studies. DSA, especially 2D-DSA, remains the gold standard for definitive diagnosis and therapeutic intervention; however, it carries a complication rate of 1.3–2.4%, with approximately 0.5% of patients experiencing potentially permanent deficits. Due to its invasiveness and associated risks, DSA is best reserved for selected cases requiring diagnostic confirmation or resistant to medical therapy.

A, Preoperative maximum intensity projection (MIP) image from a CTA study demonstrates an anterior communicating artery aneurysm (arrow). B, Postoperative CTA shows successful clipping of the aneurysm and narrowing of the anterior cerebral artery (arrow), consistent with vasospasm.

C, Digital subtraction angiography (DSA), in an anteroposterior view, confirms vasospasm involving the A1 segment of the anterior cerebral

artery. Advanced neuroimaging techniques, such as perfusion-weighted MRI (pw-MRI), have demonstrated utility in identifying discrete areas of early ischemic damage indicative of vasospasm. Diffusion-weighted MRI further contributes by revealing reductions in relative cerebral blood volume (CBV) and mean transit time, thereby assisting in the identification of candidates for triple-H therapy. Historically, MRA was primarily employed for diagnosing intracranial aneurysms and atherosclerosis-related vascular stenosis. However, its application in vasospasm diagnosis became more widespread after 1995. Modern MRA protocols utilize flow-independent strategies and offer the dual advantage of assessing radiographic vessel narrowing and providing physiological insights into vasospasm-related changes.

The therapeutic strategy for managing vasospasm extends beyond pharmacological interventions to include non-pharmacological methods that target the root causes of the condition. Central to this approach is hemodynamic optimization, which focuses on maintaining euvolemia and employing hypervolemic therapy to ensure sufficient blood volume and cerebral perfusion pressure. Additionally, blood pressure modulation through the controlled elevation of systolic levels may be utilized to enhance cerebral blood flow. In critical scenarios, endovascular cerebral angioplasty serves as an effective intervention to dilate constricted arterial segments and reestablish normal circulation. The drainage of CSF via EVD plays a pivotal role in alleviating hydrocephalus, lowering intracranial pressure, and potentially reducing vasospasm severity. Continuous monitoring of neurological function is indispensable for informing clinical decision-making and addressing potential complications. For refractory cases, decompressive craniectomy may be considered to mitigate intracranial hypertension and improve cerebral perfusion. A multidisciplinary framework that integrates both pharmacological and non-pharmacological modalities is vital for achieving optimal patient outcomes and effectively managing this intricate condition.

Management

The 2023 AHA/ASA guidelines for managing patients with SAH advocate the use of nimodipine, a calcium channel blocker (CCB) that has demonstrated efficacy in reducing DCI. Additionally, the guidelines emphasize the importance of maintaining optimal cerebral perfusion pressure and euvolemic conditions to ensure sufficient cerebral blood flow and mitigate complications associated with reduced perfusion.

In patients with aSAH, management begins with oral nimodipine (Class 1*) and regular neurological examinations (Class 1†). If the neurological exam is reliable, transcranial Doppler (TCD) monitoring is recommended (Class 2a‡); if unreliable, TCD monitoring with continuous EEG (cEEG) should be considered (Class 2a‡). Invasive multimodality neuromonitoring may be an option (Class 2b‡), but prophylactic hemodynamic augmentation should be avoided (Class 3: Harm*). If neurological status worsens or monitoring indicates decline when exams are unreliable, clinicians should address confounders by elevating blood pressure, ensuring euvolemia, and obtaining further imaging as needed (Class 2a*). If the patient improves, monitoring continues (Class 1†); if not, endovascular rescue therapy with intra-arterial spasmolytic agents with or without angioplasty may be pursued (Class 2b*). Improvement after intervention leads back to continued monitoring, while persistent deficits prompt further imaging to determine DCI or another cause for the neurological decline.

Nimodipine is the cornerstone pharmacological treatment for vasospasm following SAH, administered orally at 60 mg every four hours for 21 days to reduce both the incidence and severity of vasospasm. In acute settings, intra-arterial vasodilators such as nicardipine and papaverine are delivered via catheterization for localized arterial relief, while clazosentan, an endothelin-1 receptor antagonist, serves as an alternative by inhibiting vasoconstriction. Although not universally adopted, agents like statins and magnesium sulfate have been explored for their endothelial-stabilizing and neuroprotective effects. Non-pharmacological

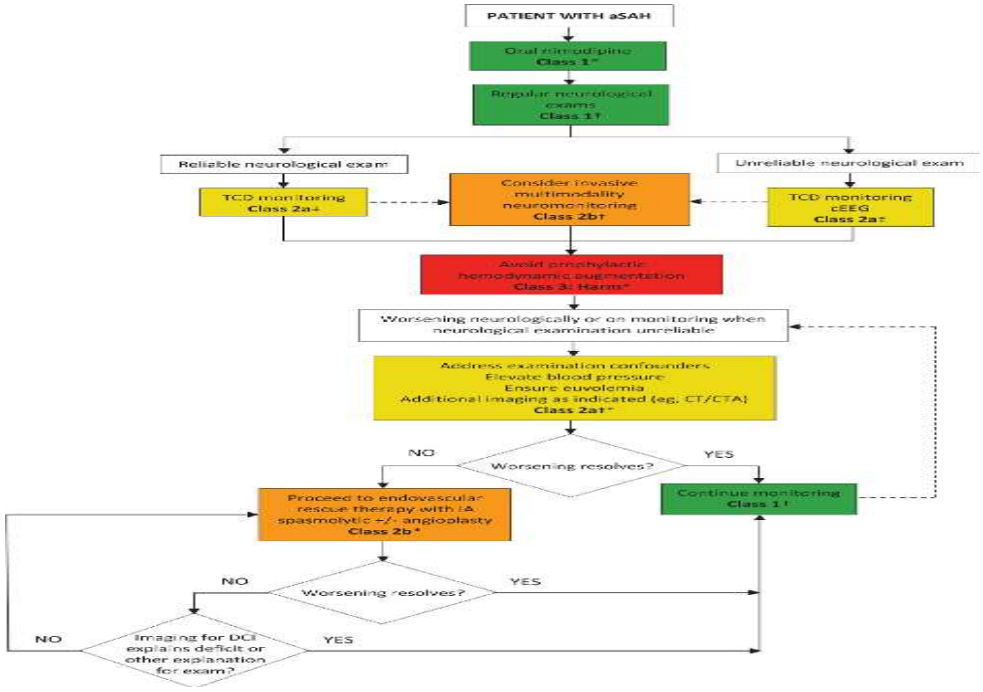

Figure 3. Management of Cerebral Vasospasm and DCI in Patients with aSAH as Outlined by the AHA/ASA

Table 2. Description of Management Approaches for Patients with Cerebral Vasospasm

Aspect	Approach	Description and Key Strategies
Pharmacological Approach	Nimodipine, Nicardipine, Papaverine, Clazosentan, Statins, Magnesium Sulfate	Nimodipine as the primary treatment, Nicardipine and Papaverine for acute management, Clazosentan as an endothelin antagonist, with Statins and Magnesium Sulfate providing additional benefits
Non-Pharmacological Approach	Hemodynamic Management, Cerebral Angioplasty, CSF Drainage, Decompressive Craniectomy	Maintaining euvolemia, hypervolemic therapy, controlled blood pressure, cerebral angioplasty, CSF drainage, and decompressive craniectomy to regulate pressure and blood flow
Triple-H Therapy	Hypervolemia, Hypertension, Hemodilution	Increasing blood volume and pressure while reducing blood viscosity to improve circulation and prevent ischemia
Advanced Techniques	Intra-Aortic Balloon Counterpulsation (IABP), Endovascular Vasospasm Reversal, Intravenous Milrinone	IABP for hemodynamic support, endovascular angioplasty, and intra-arterial vasodilator administration to manage severe vasospasm
Timing Strategy	Golden Period (3–5 Days), Early vs. Late Treatment	Early treatment within the first 3–5 days is crucial to reduce the severity of delayed cerebral ischemia (DCI); late treatment is less effective and carries a higher risk of complications
Multidisciplinary Approach	Combined Pharmacological and Non-Pharmacological Strategies, Personalized Care Plans	Integrating pharmacological and non-pharmacological approaches with routine monitoring and personalized treatment plans for optimal outcomes

approaches focus on hemodynamic optimization through euvolemia and induced hypertension to preserve cerebral perfusion, and in severe cases, endovascular interventions such as angioplasty may be employed to mechanically restore blood flow. Induced hypertension remains the mainstay hemodynamic strategy for refractory cases to augment cerebral perfusion, whereas routine hypervolemia and hemodilution, previously included in the so-called 'Triple-H therapy,' are no longer recommended due to unfavorable risk–benefit profiles. IABP is also used in refractory cases to augment systemic and cerebral circulation by enhancing diastolic pressure.

Endovascular approaches, including angioplasty and intra-arterial vasodilator infusion, are indicated in severe or refractory vasospasm, with balloon dilation restoring vessel caliber and direct drug delivery improving localized perfusion. IV milrinone, a phosphodiesterase III inhibitor, is another option under investigation for its dual role in increasing cardiac output and reducing cerebral vasoconstriction. Optimal treatment depends on timely intervention, ideally within the 3–5-day post-SAH "golden period" when the risk of DCI peaks and response to therapy is greatest. Delayed interventions beyond this window are often less effective, as irreversible cerebral injury may have already occurred, correlating with worse outcomes and higher morbidity and mortality. To improve prognosis, continuous clinical monitoring and early imaging diagnostics are essential for prompt detection and implementation of individualized treatment strategies.

Conclusion

Cerebral vasospasm is a severe and potentially life-threatening complication commonly observed following SAH, characterized by sustained constriction of cerebral blood vessels that compromises cerebral blood flow and may progress to DCI, which, if untreated, can cause profound neurological deficits or mortality. Key risk factors include the extent of blood accumulation in the subarachnoid space, the presence of hydrocephalus, demographic variables such as age and sex, and comorbidities like hypertension and smoking, with clinical manifestations ranging from severe headaches and focal neurological deficits to altered mental status, seizures, or cognitive dysfunction in advanced cases. Management integrates pharmacological and non-pharmacological approaches, with nimodipine serving as the cornerstone pharmacotherapy to prevent vasospasm and reduce morbidity, while acute interventions may involve intra-arterial vasodilators such as nicardipine or papaverine, and endothelin receptor antagonists like clazosentan to counteract vasoconstriction. Non-pharmacological strategies focus on optimizing hemodynamics through euvolemic management, controlled blood pressure modulation, and advanced interventions including endovascular angioplasty or cerebrospinal fluid drainage to relieve intracranial pressure. Preventive efforts rely on strict hemodynamic control, timely prophylactic pharmacotherapy, and advanced monitoring with modalities such as TCD and CTA to detect early vasospasm, as early intervention in the initial days post-SAH is crucial to improving clinical outcomes and minimizing long-term neurological sequelae, thereby enhancing recovery and mitigating the burden of this complex condition.

References

1. Lominadze G, Lessen S, Keene A. Vasospasm risk in surgical ICU patients with grade I subarachnoid hemorrhage. Neurohospitalist. 2016 Jan;6(1):20–3.

2. Romenskaya T, Longhitano Y, Piccolella F, Berger JM, Artico M, Taurone S, et al. Cerebral vasospasm: practical review of diagnosis and management. RRCT. 2023 Feb;18(1):12–8.

3. Aoki T, Frösen J, Fukuda M, Bando K, Shioi G, Tsuji K, et al. Prostaglandin E2 –EP2–NF-κB signaling in macrophages as a potential therapeutic target for intracranial aneurysms. Sci Signal. 2017 Feb 7;10(465):eaah6037.

4. D'Souza S. Aneurysmal subarachnoid hemorrhage. J Neurosurg Anesthesiol. 2015 July;27(3):222–40.

5. Viderman D, Tapinova K, Abdildin YG. Mechanisms of cerebral vasospasm and cerebral ischaemia in subarachnoid haemorrhage. Clin Physio Funct Imaging. 2023 Jan;43(1):1–9.

6. Romoli M, Giammello F, Mosconi MG, De Mase A, De Marco G, Digiovanni A, et al. Immunological profile of vasospasm after subarachnoid hemorrhage. IJMS. 2023 May 16;24(10):8856.

7. Zangi M, Asadi AM, Amirdosara M, Mokhtari M, Goharani R, Sanei MS, et al. Enhanced recovery after surgery (ERAS) protocol for craniotomy patients: a systematic review. Anesth Pain Med. 2024 Oct;14(5):e146811.

8. Deshaies EM, Boulos AS, Drazin D, Popp AJ. Evidence-based pharmacotherapy for cerebral vasospasm. Neurological Research. 2009 July;31(6):615–20.

9. Hasegawa S, Hasegawa Y, Miura M. Current therapeutic drugs against cerebral vasospasm after subarachnoid hemorrhage: a comprehensive review of basic and clinical studies. CDD [Internet]. 2017 Sept 6 [cited 2025 Sept 22];14(6). Available from: http://www.eurekaselect.com/144587/article

10. Siasios I, Kapsalaki EZ, Fountas KN. Cerebral vasospasm pharmacological treatment: an update. Neurolog Res Int. 2013;2013:1–20.

11. Ciurea AV, Palade C, Voinescu D, Nica DA. Subarachnoid hemorrhage and cerebral vasospasm - literature review. J Med Life. 2013 June 15;6(2):120–5.

12. Harrod CG, Bendok BR, Batjer HH. Prediction of cerebral vasospasm in patients presenting with aneurysmal subarachnoid hemorrhage: a review. Neurosurgery. 2005 Apr 1;56(4):633–54.

13. Chan AY, Choi EH, Yuki I, Suzuki S, Golshani K, Chen JW, et al. Cerebral vasospasm after subarachnoid hemorrhage: developing treatments. Brain Hemorrhages. 2021 Mar;2(1):15–23.

14. Carr KR, Zuckerman SL, Mocco J. Inflammation, cerebral vasospasm, and evolving theories of delayed cerebral ischemia. Neurol Res Int. 2013;2013:506584.

15. Wojak JC, Abruzzo TA, Bello JA, Blackham KA, Hirsch JA, Jayaraman MV, et al. Quality improvement guidelines for adult diagnostic cervicocerebral angiography: update cooperative study between the Society of Interventional Radiology (SIR), American Society of Neuroradiology (ASNR), and Society of NeuroInterventional Surgery (SNIS). J Vasc Inter Radiolog. 2015 Nov;26(11):1596–608.

16. Macdonald RL. Origins of the concept of vasospasm. Stroke [Internet]. 2016 Jan [cited 2025 Sept 23];47(1). Available from: https://www.ahajournals.org/doi/10.1161/STROKEAHA.114.006498

17. Inagawa T, Yahara K, Ohbayashi N. Risk factors associated with cerebral vasospasm following aneurysmal subarachnoid hemorrhage. Neurol Med Chir (Tokyo). 2014 June 17;54(6):465–73.

18. Rumalla K, Lin M, Ding L, Gaddis M, Giannotta SL, Attenello FJ, et al. Risk factors for cerebral vasospasm in aneurysmal subarachnoid hemorrhage: a population-based study of 8346 patients. World Neurosurgery. 2021;145:e233–41.

19. Baggott CD, Aagaard-Kienitz B. Cerebral vasospasm. Neurosurgery Clinics of North America. 2014 July;25(3):497–528.

20. Schneider UC, Xu R, Vajkoczy P. Inflammatory events following subarachnoid hemorrhage (SAH). CN. 2018;16(9):1385–95.