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Overcoming vasospasm. Timely chemical angioplasty in ruptured right posterior communicating aneurysm

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Keywords
chemical angioplasty, vasospasm management, milrinone infusion, endovascular treatment, angiographic monitoring, aneurysm embolization

ABSTRACT

This article presents a case report detailing the endovascular management of severe vasospasm in a 55-year-old patient following the rupture of a right posterior communicating aneurysm. Initially presenting with a Fisher grade 4 subarachnoid haemorrhage, the patient underwent embolization treatment, which resulted in good condition. However, on the 9th day, the patient experienced a sudden deterioration, marked by severe vasospasm and neurological deficits.

Diagnostic procedures, including cerebral angiography, revealed extensive vasospasm affecting multiple cerebral arteries. In response, endovascular therapy was initiated, consisting of the intra-arterial administration of Milrinone and Nimodipine. This intervention aimed to alleviate vasospasm and enhance cerebral perfusion. Angiographic imaging post-intervention demonstrated significant improvement in vascular perfusion, correlating with a rapid neurological response. Notably, the patient exhibited immediate improvement in motor deficits and dysarthria following treatment.

This case underscores the critical importance of timely recognition and intervention in managing complications post-aneurysmal rupture, particularly severe vasospasm, which poses significant risks of neurological sequelae. The successful application of endovascular techniques, including chemical angioplasty, highlights the evolving landscape of neurointerventional procedures in addressing complex cerebrovascular pathology.

The case further emphasizes the necessity of a multidisciplinary approach, involving collaboration between neurosurgery, interventional radiology, and neurocritical care, to optimize patient outcomes in cerebrovascular emergencies. Early detection, prompt intervention, and vigilant post-procedural monitoring remain pivotal in mitigating the risks associated with cerebral aneurysm rupture and its sequelae.

1. INTRODUCTION

Vasospasm following ruptured brain aneurysms represents a pivotal challenge in neurocritical care, significantly affecting outcomes by predisposing patients to delayed cerebral ischemia. This condition

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underscores the necessity of a nuanced understanding of cerebral vasculature responses post-rupture and the strategic application of interventions to prevent ischemic complications. The literature emphasizes the imperative of timely and aggressive management strategies to improve patient prognosis, focusing on the initial stabilization of life-threatening conditions, meticulous control of blood pressure, maintenance of fluid and electrolyte balance, and the prophylactic administration of nimodipine.

Early intervention is crucial, with strategies encompassing the rapid repair of the ruptured aneurysm via surgical clipping or endovascular coiling to secure the aneurysm and prevent rebleeding. Such prompt actions are supported by studies showing the correlation between the timing of treatment post-rupture and the incidence of complications such as rebleeding, cerebral infarcts, and mortality. Specifically, treatments administered within the critical first hours after rupture can significantly reduce the risk of subsequent vascular events, highlighting the importance of early diagnosis and intervention.

Beyond the initial surgical interventions, the management of vasospasm involves a sophisticated regimen of pharmacological agents aimed at vasodilation to restore adequate cerebral blood flow. Nimodipine, a calcium channel blocker, is widely recognized for its efficacy in preventing vasospasm and improving neurological outcomes. Its role is augmented by the selective use of intraarterial vasodilators, including papaverine, nicardipine, and verapamil, particularly in refractory cases where conventional treatments fail to alleviate the vasospasm.

The utilization of vasodilators extends to a diverse array of agents, each with specific mechanisms of action tailored to counteract the pathological constriction of cerebral vessels. Among these, magnesium sulfate acts as a non-selective calcium channel blocker and has neuroprotective properties. Clazosentan, an endothelin receptor antagonist, and milrinone, a phosphodiesterase III inhibitor, represent newer therapeutic avenues being explored for their potential to reduce vasospasm-induced complications in clinical trials.

The literature review further explores chemical angioplasty with milrinone and nimodipine as an innovative treatment modality. This approach targets the underlying pathophysiological mechanisms of vasospasm, offering a multimodal strategy to enhance cerebral vasodilation and blood flow. Clinical studies have documented the effectiveness of this therapy in reducing vasospasm severity and improving neurological outcomes, suggesting its value as a complementary treatment in the neurocritical care arsenal.

The study by Labeyrie et al. on distal balloon angioplasty emphasizes its efficacy in decreasing the risk of delayed cerebral infarction, highlighting the importance of endovascular interventions. Bashir et al.'s work on intra-arterial nimodipine points to its beneficial influence on clinical outcomes, reinforcing the role of targeted pharmacological therapies. These references, along with foundational research like Aaslid et al.'s evaluation of cerebrovascular spasm with Doppler ultrasound and Allen GS et al.'s trial on nimodipine, underscore the multifaceted approach required to manage vasospasm, combining diagnostic precision, prophylactic pharmacotherapy, and innovative endovascular techniques to improve patient outcomes. Integrating these findings into the management strategy provides a comprehensive and evidence-backed framework for treating vasospasm after brain aneurysm rupture.

Safety considerations are paramount, with the literature indicating that while these pharmacological interventions are generally well-tolerated, careful monitoring for potential adverse effects, such as hypotension, is essential. The goal is to achieve a delicate balance between therapeutic efficacy and safety, optimizing dosing and administration protocols to maximize patient benefits while minimizing risks.

Future research directions are poised to refine our understanding of vasospasm management, with ongoing investigations into the optimal timing, duration, and patient selection criteria for these interventions. The evolution of treatment protocols, informed by robust clinical evidence, promises to enhance the quality of care for patients suffering from the aftermath of ruptured brain aneurysms.

In sum, the management of vasospasm following ruptured brain aneurysms encapsulates a multidisciplinary effort, integrating surgical, pharmacological, and supportive strategies to mitigate the risk of delayed cerebral ischemia and optimize patient outcomes. Through the judicious

application of established and emerging therapies, the neurocritical care community continues to advance the frontier of care for this complex and challenging condition.

2. Materials and methods

Patient underwent comprehensive diagnostic assessments upon admission to evaluate their neurological status and identify underlying vascular abnormalities. The diagnostic protocol involved cerebral angiography to visualize the vascular anatomy and embolization of the ruptured posterior communicating aneurysm in the first case and severe vasospasm following meningitis hemorrhage in the second case.

Therapeutic interventions involved meticulous preparation for endovascular procedures, including femoral artery puncture and the insertion of catheters and guides. In the first procedure, platinum coil embolization via a coaxial system occluded the aneurysm, followed by angiographic confirmation. In the second procedure, intra-arterial chemical angioplasty with Milrinone and Nimodipine alleviated severe vasospasm, monitored through angiographic imaging. Post-procedural care encompassed manual femoral artery closure, compressive dressing, and cerebral CT scans for outcome assessment and complication detection. Patients received intravenous Heparin bolus during procedures to prevent thrombotic events.

3. Case report

3.1. Presentation in ED and embolization

A 55-year-old female known to have hypertension, type 2 diabetes mellitus, presented to the emergency department with a Fisher 4 subarachnoid hemorrhage caused by rupture of the right posterior communicating aneurysm. At admission, the patient has a GCS of 15 without neurological deficits.

The patient was transferred to the interventional neuroradiology unit, where cerebral angiography with digital subtraction (DSA) was performed, which revealed a ruptured giant posterior communicating aneurysm. Embolization of the aneurysm was performed with platinum coils, the intervention proceeded without complications. Post-procedurally, the woman woke up without neurological deficits, with normal cardiovascular values. Vasospasm prevention therapy with Nimodipine was initiated.

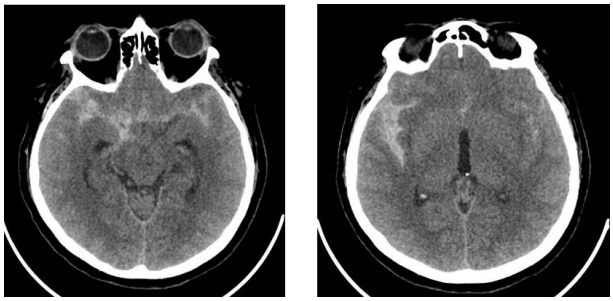


Figure 1. Cranio-cerebral native CT scan in the ED (a-b) subarachnoid haemorrhage with quantitatively greater extension at the level of the right sylvian fissure.

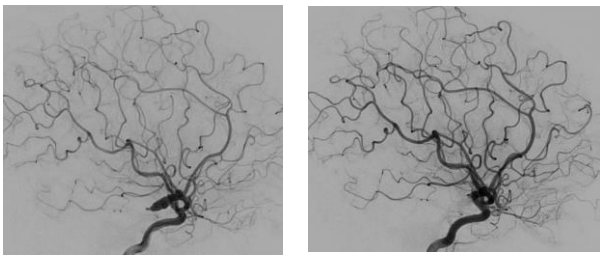


Figure 2. Digital subtraction angiogram (DSA) in lateral view of the right internal carotid artery: (a) Giant posterior communicating saccular aneurysm; (b) DSA control after endovascular embolization with platinum coils.

3.2 Clinical deterioration on the 9th day

In the morning of the 9th day, the patient shows mild fatigue. An interdisciplinary consultation is requested for a complete neurological examination which is normal, without changes. A native cranio-cerebral CT scan is performed, which reveals a small hypodense area at the right frontal sylvian level (Figure 3a). A few hours later, the patient presents, with progressive worsening, left hemiparesis, up to hemiplegia and dysarthria. An emergency cranio-cerebral MRI examination is performed, which highlights an ischemic area in the territory of the right middle cerebral artery (Figure 3b).

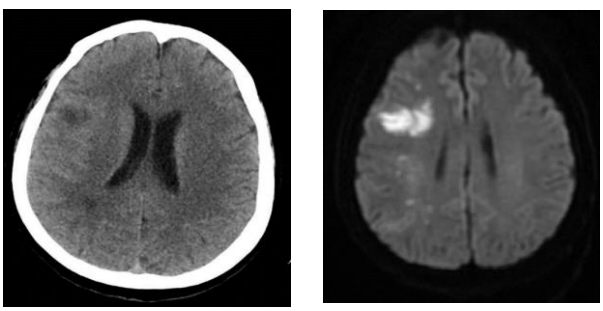
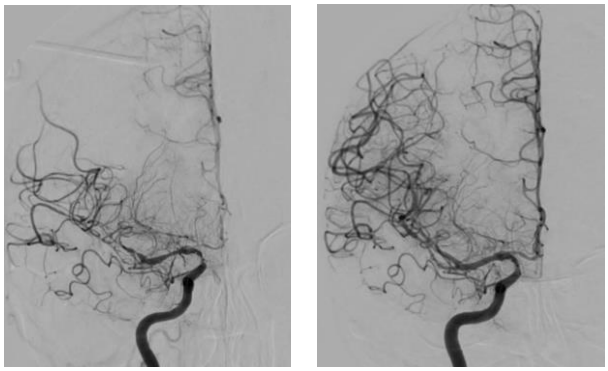


Figure 3. Brain imagistic investigations at the time of first neurological symptoms onset: (a) Cranial CT scan performed as

routine - the patient complains of mild fatigability; (b) Brain MRI performed after the onset of left hemiplegia and dysarthria.

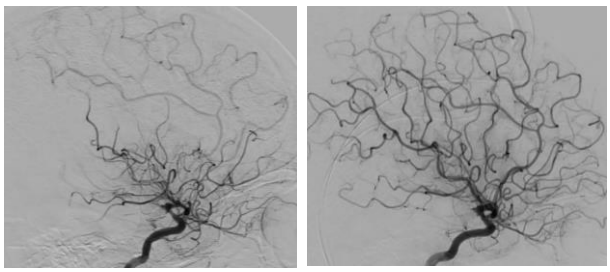
3.3 Chemical angioplasty with Milrinone and Nimodipine for vasospasm therapy

DSA highlighted a severe vasospasm at the level of the right carotid termination, right A1, segment M1 and M2 of the right Sylvian artery. The right sylvian branches have a filiform appearance with very delayed parenchymography.



(a) (b)

Figure 4. DSA in frontal view of the right internal carotid artery: (a) Severe vasospasm at the level of the right carotid termination, right A1, segment M1 and M2; (b) After the selective intra-arterial injection of vasodilators, a reduction in vascular spasm is observed with a net improvement of cerebral perfusion.



(a) (b)

Figure 5. DSA in lateral view of the right internal carotid artery: (a) Severe vasospasm at the level of the right carotid termination, right A1, segment M1 and M2; (b) After the selective intra-arterial injection of vasodilators, a reduction in vascular spasm is observed with a net improvement of cerebral perfusion.

4. Discussion

The efficacy of chemical angioplasty using Milrinone and Nimodipine in managing severe vasospasm following the rupture of a posterior communicating aneurysm has been a subject of significant research interest. It is imperative to analyze how these medications act synergistically to induce cerebral vasodilation and improve cerebral blood flow, leading to rapid symptom relief and enhanced neurological recovery.

Timely recognition and intervention are paramount in managing complications post-aneurysmal rupture, particularly severe vasospasm. Long-term neurological outcomes and follow-up strategies for patients undergoing chemical angioplasty require thorough evaluation. Continued surveillance and monitoring, along with the potential role of advanced imaging modalities and functional assessments, are crucial in detecting and managing any late complications or recurrence of vasospasm.

A multidisciplinary approach involving neurosurgery, interventional radiology, and neurocritical care is essential for optimizing patient outcomes. As highlighted by our example, a coordinated multidisciplinary approach enables comprehensive evaluation, timely intervention, and vigilant post-procedural monitoring, ultimately enhancing the quality of care and patient safety.

Comparing chemical angioplasty to traditional management strategies raises important considerations regarding efficacy, safety, and patient outcomes. While Milrinone and Nimodipine have shown promise in reducing the incidence of delayed cerebral ischemia and facilitating functional recovery, rigorous comparative studies are essential. These studies would offer clearer insights into the benefits of chemical angioplasty over standard treatments, including the use of oral or intravenous vasodilators and the implementation of Triple-H therapy (hypertension, hypervolemia, and hemodilution).

Considering the generalizability of findings from case reports to broader clinical practice is essential. Proposing avenues for future research, including larger prospective studies and randomized controlled trials, can further elucidate the optimal timing, dosing, and patient selection criteria for chemical angioplasty in managing vasospasm after brain aneurysm rupture.

Exploring patient-specific factors that may influence the response to chemical angioplasty is necessary for personalized treatment strategies. Additionally, addressing ethical and legal considerations, enhancing patient education and counseling, and understanding the mechanisms of action of Milrinone and Nimodipine are vital aspects of optimizing vasospasm management.

The exploration of genetic and molecular biomarkers associated with vasospasm susceptibility represents an exciting frontier in neurocritical care. Such advancements could lead to tailored treatment strategies that account for individual variability in response to therapy. Additionally, addressing disparities in treatment outcomes across different patient populations could further enhance the effectiveness and equity of vasospasm management.

5. Conclusion

This case underscores the critical need for timely intervention in managing complications post-aneurysmal rupture, particularly severe vasospasm. Chemical angioplasty with Milrinone and Nimodipine led to significant improvements in vascular perfusion and neurological deficits, reflecting advancements in neurointerventional procedures. A multidisciplinary approach involving neurosurgery, interventional radiology, and neurocritical care proved vital in optimizing patient outcomes. Early detection, swift intervention, and vigilant post-procedural monitoring are essential for mitigating risks associated with cerebral aneurysm rupture and its sequelae.

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