From Vasospasm to Ventilator: Milrinone-Related High-Output Heart Failure and ARDS

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Introduction

Milrinone is increasingly utilized as a therapeutic option for cerebral vasospasm and delayed cerebral ischemia (DCI) following aneurysmal subarachnoid hemorrhage (SAH). Evidence suggests milrinone may improve cerebral perfusion and reduce the need for vasopressors and endovascular interventions.

While generally well tolerated, milrinone is a phosphodiesterase-3 inhibitor with potent vasodilatory and positive inotropic effects. Excessive systemic vasodilation can precipitate high-output heart failure and respiratory complications — a mechanism that is underrecognized in the neurocritical care setting.

We present a case of high-output heart failure and severe ARDS precipitated by continuous intravenous milrinone therapy for cerebral vasospasm following SAH.

Case Description

A 57-year-old woman presented with sudden-onset headache and loss of consciousness. CT imaging revealed diffuse subarachnoid hemorrhage and multiple intracranial aneurysms. She underwent right MCA aneurysm embolization.

CTA demonstrated severe cerebral vasospasm. Continuous IV milrinone was initiated with norepinephrine co-infusion to maintain cerebral perfusion pressure.

Shortly after milrinone initiation, the patient developed progressive hypoxemia and pulmonary edema refractory to aggressive diuresis. Echocardiography demonstrated hyperdynamic left ventricular function with markedly elevated cardiac output, consistent with high-output heart failure.

Rapid progression to severe ARDS ensued, refractory to conventional ventilation. Milrinone was discontinued and targeted hemodynamic support initiated. The patient required neuromuscular blockade and airway pressure release ventilation (APRV) for oxygenation optimization.

Over the following days, oxygenation improved, paralytics were weaned, and cardiac output normalized following cessation of milrinone. The patient ultimately recovered sufficient respiratory function to transition to conventional ventilation and was successfully extubated.

Discussion & Conclusion

Discussion

This case highlights a rare but serious complication of milrinone therapy in the neurocritical care setting. The combination of systemic vasodilation, markedly elevated cardiac output, and resultant capillary leak likely contributed to pulmonary edema and severe hypoxemia meeting ARDS criteria.

Recognition of the high-output physiology was essential: standard diuresis alone was insufficient. Discontinuation of the offending agent combined with advanced ventilatory strategies — APRV and neuromuscular blockade — proved lifesaving.

Conclusion

Milrinone therapy for cerebral vasospasm following SAH can trigger high-output heart failure and contribute to severe respiratory failure meeting ARDS criteria. Clinicians should maintain a high index of suspicion for cardiogenic contributions to respiratory failure in SAH patients receiving milrinone, facilitating early recognition and intervention.