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Case Report

Intravenous Milrinone for Hemodynamic Rescue in Spontaneous Intracerebral Hemorrhage with Intraventricular Extension

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ABSTRACT

Spontaneous intracerebral hemorrhage (ICH) with intraventricular extension (IVH) is a life-threatening condition associated with high rates of morbidity and mortality. Current management is primarily supportive, with few pharmacologic options available to optimize cerebral perfusion. Milrinone, a phosphodiesterase-3 (PDE-3) inhibitor commonly used in cardiology to boost cardiac output and stabilize hemodynamics, has rarely been explored in neurocritical care. We describe a 70-year-old man with hypertension who experienced a sudden collapse and left-sided paralysis. Imaging revealed a large hemorrhage affecting the right thalamus and basal ganglia with extension into the ventricles. Despite standard interventions, his neurological condition deteriorated. Intravenous milrinone was initiated as a salvage therapy to support mean arterial pressure (MAP) and preserve cerebral perfusion pressure (CPP). Following treatment, his hemodynamics stabilized, intracranial pressure remained controlled, and neurological function improved. At discharge, he had a Glasgow Coma Scale (GCS) of 14, and follow-up imaging confirmed complete resolution of the clot. This case highlights the potential of milrinone to support both systemic and cerebral hemodynamics in patients with ICH/IVH, demonstrating its combined cardiac and neuroprotective effects. To our knowledge, this is among the first documented uses of intravenous milrinone in spontaneous hemorrhagic stroke, suggesting a promising new application for PDE-3 inhibition in critical care beyond traditional cardiology.

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Introduction

Spontaneous intracerebral hemorrhage (ICH) is one of the life-threatening forms of stroke, with a high morbidity and mortality rate of about 30% to 40% [1-2]. It is characterized by brain damage resulting from the sudden leakage of blood into the brain tissue due to a ruptured cerebral blood vessel [2-3]. Additionally, intraventricular hemorrhage (IVH) poses an extra clinical challenge in approximately 40% to 45% of these cases [4]. Several risk factors have been identified for these major neurological events, including age, smoking, hypertension, and hypercholesterolemia [4-5]. Despite advances in acute neurocritical care, management remains largely supportive, focusing on rapid blood pressure control, reversal of coagulopathies, intracranial pressure management, and treatment of hydrocephalus [6]. Multiple long-term complications can arise following ICH or IVH, including re-bleeding, ischemia, inflammation, and an increase in hematoma size. Currently, there are no effective treatments available to mitigate these consequences, improve prognosis, or reduce morbidity and mortality in affected patients [7-12]. Milrinone, a phosphodiesterase-3 (PDE-3) inhibitor, was initially developed in the late 1980s as an intravenous and oral therapy for heart failure, but the oral form was abandoned after trials such as the PROMISE study revealed increased mortality and arrhythmia risk in chronic heart failure [13]. More recently, however, the role of milrinone has extended beyond cardiology into neurology, particularly in subarachnoid hemorrhage (SAH) [14], where it has demonstrated vasodilatory, hemodynamic, and neuroprotective effects. By augmenting cardiac output through PDE-3–mediated inotropy, milrinone helps maintain mean arterial pressure even in the presence of elevated intracranial pressure, thereby preserving cerebral perfusion pressure (CPP) = mean arterial pressure (MAP) – intracranial pressure (ICP), a critical determinant of cerebral blood flow [15-17]. Simultaneously, its direct vasodilatory effect on cerebral resistance vessels enhances regional microcirculation and mitigates ischemia in perihematomal tissue.

These combined actions, well established in SAH, may explain the clinical stabilization observed in our patient with spontaneous ICH/IVH [14].

Here, we present a case of spontaneous intracerebral hemorrhage with intraventricular extension in which intravenous milrinone was administered as salvage therapy.

To the best of our knowledge, this is among the first reported uses of milrinone in this setting, underscoring its potential role in managing hemodynamic compromise in patients with hemorrhagic stroke. Although traditionally recognized as a cardiac agent, milrinone is increasingly regarded as having broader applications beyond cardiology, including the optimization of cerebral perfusion in neurocritical care and the preservation of vascular perfusion pressure.

Case Report

A 70-year-old man with a history of hypertension (on losartan 80 mg daily) and no history of coagulopathy, smoking, or alcohol use, suddenly collapsed in the street.

Upon arrival at the emergency department, his Glasgow Coma Scale (GCS) score was 12 (E4V2M6), reflecting moderate impairment but preserved brainstem reflexes. Neurological examination revealed left hemiplegia, left facial palsy, disorientation, and incomprehensible speech, although eye contact and the ability to follow simple motor commands were retained. Vital signs were stable: blood pressure 140/70 mmHg, heart rate 77 bpm, respiratory rate 17/min, oxygen saturation 96% on room air, and blood glucose 130 mg/dL.

Investigations

Neuroimaging with non-contrast computed tomography (CT) revealed a large hyperdense hemorrhage involving the right thalamus, basal ganglia, corona radiata, centrum semiovale, and cerebral peduncle, accompanied by surrounding edema, a 3-mm leftward midline shift, and intraventricular extension into the lateral and third ventricles (Figure 1). The ECG demonstrated sinus rhythm without evidence of acute ischemic changes. Chest X-ray showed a normal cardiac silhouette with clear lung fields. Abdominal radiographs in both supine and erect positions revealed normal renal outlines, no stones, and an unremarkable bowel gas pattern. Laboratory investigations indicated hemoglobin of 13.6 g/dL, platelet count of 225×10^9 /L, INR of 1.0, creatinine of 0.9 mg/dL, sodium of 138 mmol/L, potassium of 4.2 mmol/L, and magnesium of 1.9 mg/dL. CT angiography/ magnetic resonance angiography was not performed due to hemodynamic instability and institutional policy favoring stabilization before advanced vascular imaging.

Clinical Course and Interventions

The patient was admitted to the intensive care unit (ICU), where he was intubated and treated with osmotherapy for ICP control, strict blood pressure regulation, seizure monitoring (not observed), and mechanical ventilation.

Despite these measures, neurological status worsened between ICU days 7 and 18, with imaging findings suggesting reduced cerebral perfusion (Figure 2). In an effort to preserve CPP, intravenous milrinone was introduced as salvage therapy according to the following protocol (Table 1).

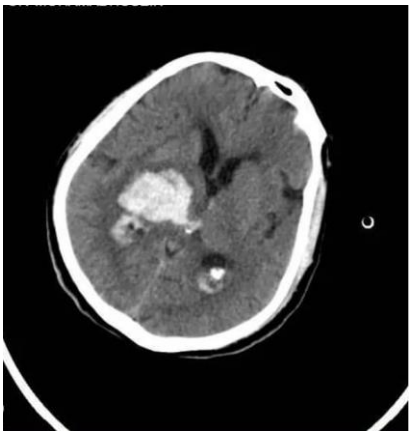

Figure 1- Non-contrast CT on admission (showing extensive right-sided intracerebral hemorrhage with intraventricular extension and midline shift (hemorrhage volume: 33.7 mL))

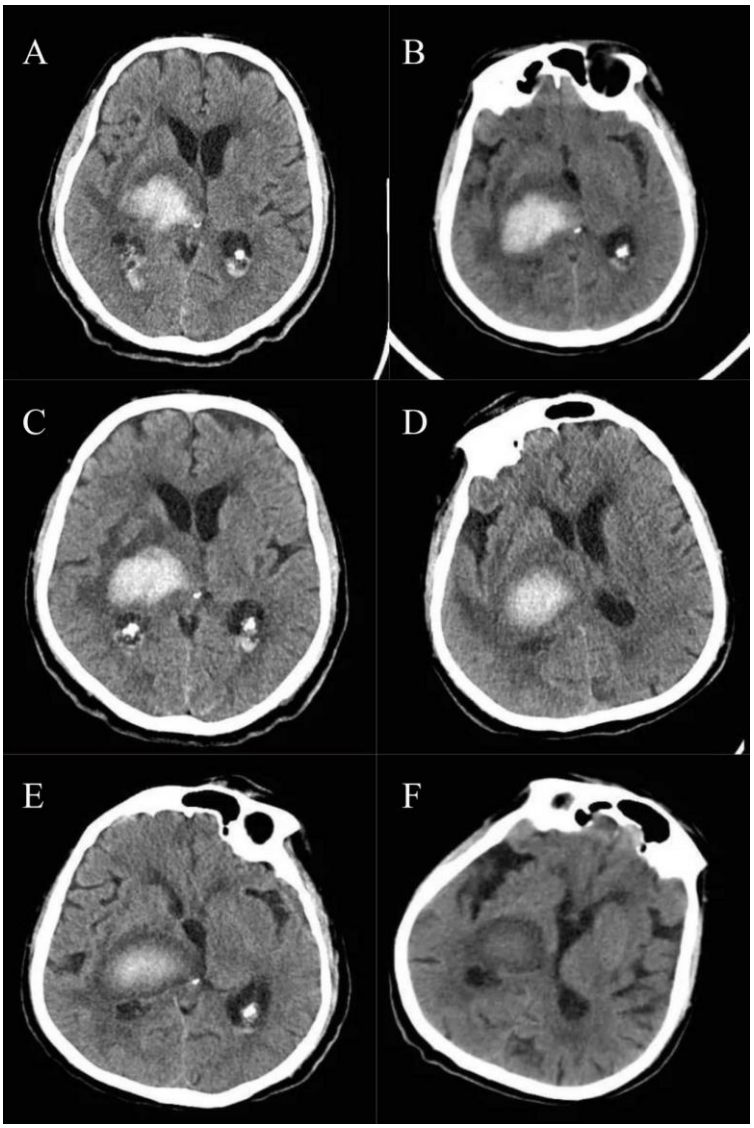

Figure 2- Follow-up imaging (ICU days 7–18) (Demonstrating reduced cerebral perfusion (A: Hemorrhage volume: 24 mL; B: 23 mL; C: 21.8 mL; D: 13.5 mL; E: 10.9 mL, F: recovered))

Table 1- Milrinone administration protocol

ICU Day	Dose / Infusion Protocol	Holding Criteria
Day 7	2 mg IV over 1 h, then 1 mg/h × 24 h	Hold if MAP < 100 mmHg
Day 9	Same as Day 8	Hold if MAP < 100 mmHg
Day 11	1 mg IV over 1 h, then 1 mg/h × 24 h	Hold if MAP < 100 mmHg
Day 18	2 mg IV over 1 h, then 1 mg/h × 24 h	Hold if MAP < 100 mmHg

MAP=Mean arterial pressure

No vasopressors were required. After milrinone initiation, the MAP increased from 82 mmHg to a sustained 95–105 mmHg, while ICP remained stable on serial monitoring. This hemodynamic response was accompanied by clinical stabilization.

However, transient hypertension, hypokalemia, hypophosphatemia, and hypomagnesemia were observed as adverse effects; all were corrected with appropriate replacement. No arrhythmias were detected during continuous ECG monitoring.

Outcome and Follow-Up

By ICU day 27, the patient was successfully extubated. At the time of ICU discharge, neurological status had improved with a GCS of 14, NIH Stroke Scale (NIHSS) score of 8, and modified Rankin Scale (mRS) score of 3. He was hemodynamically stable and transferred to the ward for continued rehabilitation. On the follow-up, the clot was completely absorbed 49 days later.

Discussion

ICH and IVH are medical emergencies with notable burdens across low- and middle-income countries [18]. Guidelines emphasize rapid stabilization and aggressive medical management in the first 72 hours. This includes strict blood pressure control to reduce the risk of hematoma expansion (e.g., targeting systolic blood pressure (SBP) ≤140 mmHg for SBP 150–220 mmHg), reversal of any coagulopathies (such as administering prothrombin complex concentrate (PCC) or vitamin K for warfarin), and intracranial pressure control [2]. Acute hydrocephalus from IVH is managed by prompt external ventricular drain (EVD) placement, which relieves ventricular pressure and prevents neurologic deterioration [19]. Seizures, if they occur, should be treated (though prophylactic antiseizures are not routinely recommended) [20]. Furthermore, daily serial CT could identify hydrocephalus, particularly amid neurological decline [21]. Despite these measures, it is important to note that no specific pharmacologic agent has been definitively proven to improve outcomes in acute ICH/IVH beyond supportive care [22]. In contrast to aneurysmal SAH, there is no established drug therapy to prevent delayed complications after primary ICH/IVH. Early complications like rebleeding, edema, or vasospasm are addressed by the general measures above, but no targeted neuroprotective drug has shown clear benefit in clinical trials. It is worth distinguishing the vasospasm and delayed ischemia seen in SAH from the typical ICH course [23]. Cerebral vasospasm leading to delayed cerebral ischemia (DCI) is a well-known phenomenon in SAH (occurring in ~30% of SAH patients), and nimodipine is routinely used in SAH to mitigate this risk [24]. However, vasospasm is not common in ICH/IVH unless there is an associated subarachnoid component [25]. Primary IVH can rarely trigger diffuse vasospasm, but only a handful of such cases have been reported. Therefore, prophylactic vasospasm treatments (like calcium channel blockers) are not part of standard ICH/IVH management. Instead, the focus in the acute phase remains on preventing hematoma expansion (through blood pressure management and hemostatic therapies) and managing intracranial hypertension or hydrocephalus (with measures like head elevation, osmotic therapy, and EVD if needed).

A wide spectrum of pharmacological treatments has been scrutinized for attenuating delayed complications, including ischemia, inflammation, and hematoma expansion in hemorrhagic stroke. Evidence, predominantly preclinical or extrapolated from SAH/ischemic paradigms, underscores their potential, albeit with caveats regarding clinical translation. (Table 2) summarizes pivotal agents, potential mechanisms, and limitations of previously studied drugs. Milrinone, a PDE-3 inhibitor, exerts vasodilatory, inotropic, and anti-inflammatory effects, predominantly validated for cerebral vasospasm and DCI in the context of SAH [27]. By increasing cyclic AMP, milrinone causes smooth muscle relaxation and vasodilation in cerebral arteries and augments cardiac output, thereby improving cerebral perfusion [17] milrinone activates protein kinase A, enhances calcium signaling, reduces oxidative stress, and modulates apoptotic and inflammatory pathways [28-30]. Preclinical and clinical studies further suggest that it may inhibit tumor-promoting signaling cascades, including phosphatidylinositol 3-kinase/protein kinase B (PI3K/AKT), mitogen-activated protein kinase (MAPK), and Wnt/β-catenin pathways, while limiting mitochondrial reactive oxygen species (ROS) production [30-31]. Clinical evidence, mostly case series and cohort studies, indicates that milrinone can reverse vasospasm angiographically and may improve neurologic outcomes after aneurysmal SAH. In fact, a systematic review found that intravenous and intra-arterial milrinone therapy was associated with the resolution of vasospasm in the majority of reported SAH cases [32].

Table 2- Pharmacological Agents Investigated for Mitigating Delayed Complications in Hemorrhagic Stroke: Mechanisms and Limitations

Drug/Category	Potential Mechanism	Evidence in Hemorrhagic Stroke	Key Limitations/Notes
Calcium Channel Blockers (e.g., Nimodipine) [12]	Vasodilation, calcium influx inhibition, BP modulation, and ICP reduction	Consolidated in SAH for DCI prophylaxis; ICH trials evince hematoma absorption and neurological amelioration, with reduced ICP and BP variability (meta-analyses, RCTs).	Hypotension risk; intraventricular formulations experimental; not ICH guideline-standard.
Magnesium Sulfate (NMDA Antagonist) [11]	Neuroprotection, hematoma expansion reduction, and anticoagulation modulation	Linked to diminished hematoma expansion and neurological decline in ICH; FAST-MAG trial evinced no broad stroke benefit, yet hypomagnesemia correction may mitigate secondary injury (RCTs, observational).	Inconsistent IVH prevention; potential hemostatic role in hypomagnesemia cohorts; requires further RCTs.
Statins [10]	Anti-inflammatory, vascular stabilization	Varied cohorts indicate reduced ICH recurrence, yet heightened risk in lobar ICH or high-dose regimens (meta-analyses, observational).	Dose-dependent hemorrhage risk; avoid in prior ICH; double-edged sword per recent reviews.
Minocycline [9]	MMP inhibition, BBB fortification	Preclinical suppression of BBB dysfunction and neuroinflammation in TBI/ICH models; vasculostatic potential for malformations (rodent studies).	Predominantly preclinical; no large ICH trials; transient ischemic risk in some cohorts.
Cholinergic Drugs (e.g., Piracetam, Citicoline) [7-8]	Neuroprotection, membrane repair, neurotransmission modulation	Acute ischemic stroke outcomes improved; combination therapy yields superior neuroprotection in ICH models, reducing deficits (RCTs, open-label studies).	Small-scale studies; not ICH guidelines; sequential/combination therapy promising.
Melatonin [26]	Antioxidant, anti-inflammatory, and edema reduction	Preclinical ICH models show neuronal damage and edema mitigation; observational studies indicate trends toward reduced mortality and disability, though not statistically significant at low doses (2-30 mg; RCTs, animal models).	Dose-dependent efficacy; higher doses (>10 mg) are safe but require RCTs; no delirium benefit.

BBB=Blood–brain barrier, BP=Blood pressure, DCI=Delayed cerebral ischemia, FAST-MAG=Field Administration of Stroke Therapy–Magnesium, ICH= Intracerebral hemorrhage, ICP=Intracranial pressure, IVH=Intraventricular hemorrhage, MMP=Matrix metalloproteinase, NMDA=N-methyl-D-aspartate, RCT=Randomized controlled trial, SAH=Subarachnoid hemorrhage, TBI=Traumatic brain injury

Milrinone is now frequently used as a salvage therapy for refractory vasospasm in many neurocritical care centers, administered either systemically or via intra-arterial/intraventricular routes when standard hypertensive therapy fails [33].

Together, these mechanisms and the evidence mentioned support the potential of milrinone as a therapeutic agent in ICH/IVH by reducing ischemic injury, oxidative damage, apoptosis, and delayed cerebral ischemia.

Recent studies have even suggested that proactive use of milrinone guided by cerebral perfusion monitoring can reduce the incidence of delayed cerebral infarctions after SAH.

A comprehensive literature scrutiny, encompassing PubMed, Google Scholar, and Scopus through 2025, reveals no antecedent reports or trials of milrinone for spontaneous ICH or IVH; all applications pertain to aneurysmal or traumatic SAH with vasospasm. Isolated instances of vasospasm in primary IVH have been managed with intra-arterial therapies, without milrinone. This paucity underscores the present case as potentially the novel documented utilization of intravenous milrinone as salvage therapy in hemodynamic-dependent ICH/IVH, averting surgical morbidity.

In summary, this case illuminates innovative deployment of milrinone for brain tissue salvage in symptomatic ICH and IVH, potentially augmenting outcomes without surgery.

Replication in expansive cohorts could refine management of reversible hemodynamic compromise in hemorrhagic stroke.

Conclusion

In conclusion, this case report indicates that intravenous milrinone could be an effective salvage treatment for patients experiencing spontaneous ICH with IVH and hemodynamic instability. By aiding in maintaining MAP, preserving cerebral blood flow, and improving microvascular circulation, milrinone played a role in stabilizing neurological function without requiring surgical intervention. Although the current evidence is limited to this case and related insights from SAH studies, these results highlight a promising therapeutic option that deserves further exploration in larger clinical trials. Prompt identification and targeted pharmacological intervention may provide a new approach to reduce secondary injury and enhance outcomes for patients at high risk of hemorrhagic stroke.

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Authors' contributions

MM: conceptualization, methodology, project administration, supervision, validation, review and editing. **FA:** formal analysis, investigation, methodology, visualization, writing original draft, review and editing. **AN:** writing original draft, visualization, literature review and editing. **SJ:** writing original draft, visualization, literature review and editing. **RS**: conceptualization, methodology, writing original draft, review and editing, **ES:** data curation, investigation, writing original draft. **AN:** project administration, supervision. **AM:** review and editing. All authors contributed equally to this work.

Ethics approval and consent to participate

This case report was conducted following the ethical principles outlined in the Declaration of Helsinki. Both the patient and his legal guardian provided written informed consent to participate and share clinical details.

Consent for publication

Written informed consent was obtained from the patient and his legal guardian for publication of this case report, including any accompanying images, ensuring their agreement to share the medical information.

Availability of data and material

Available from the corresponding author upon request.

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