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Conservative Treatment With Mannitol In A Young Adult With Intracerebral Hemorrhage Resulting In Complete Neurological Recovery: A Case Report

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Abstract

Intracerebral hemorrhage (ICH) resulting from arteriovenous malformation (AVM) rupture is a neurological emergency associated with increased intracranial pressure, cerebral edema, and a high risk of morbidity and mortality. Although surgical intervention is the standard treatment for selected patients, conservative management may be considered in clinically stable individuals. The role of mannitol in improving outcomes in young patients with AVM-related ICH remains incompletely understood. We report the case of a 22-year-old man who presented with sudden-onset severe headache accompanied by nausea and vomiting. Brain computed tomography revealed a left parieto-occipital intracerebral hemorrhage with intraventricular extension, while subsequent imaging confirmed a ruptured AVM as the underlying cause. The patient was hemodynamically stable and exhibited no significant focal neurological deficits. Conservative treatment consisting of intravenous mannitol, antihypertensive therapy, analgesics, and supportive care was initiated with close neurological monitoring. During hospitalization, the patient's clinical condition progressively improved, with resolution of headache, nausea, and vomiting, accompanied by recovery of consciousness. He was discharged without residual neurological deficits. This case highlights that conservative management with mannitol may achieve favorable neurological outcomes in carefully selected young adults with AVM-related intracerebral hemorrhage. Early diagnosis, appropriate intracranial pressure control, and close clinical monitoring are essential for optimizing patient recovery. Further studies are needed to establish the role of conservative therapy in similar clinical settings.

Keywords: Intracerebral Hemorrhage; Arteriovenous Malformation; Mannitol; Conservative Management; Intracranial Pressure

INTRODUCTION

Intracerebral hemorrhage (ICH) is a neurological emergency characterized by bleeding within the brain parenchyma that results from the rupture of intracranial blood vessels. This condition leads to hematoma formation, increased intracranial pressure (ICP), compression of adjacent brain tissue, and potentially irreversible neurological injury. ICH may occur secondary to traumatic brain injury or spontaneously due to underlying vascular abnormalities and chronic hypertension. As a subtype of hemorrhagic stroke, ICH is associated with substantial morbidity and mortality and requires prompt diagnosis and management (Dewi & Asman, 2021).

Stroke remains one of the leading causes of disability and death worldwide and represents a major public health burden (Feigin et al., 2025). According to the 2023 Indonesian Health Survey, the national prevalence of stroke reached 8.3 per 1,000 population, making it one of the leading causes of mortality and long-term disability in Indonesia. Stroke is broadly classified into ischemic and hemorrhagic subtypes, each requiring fundamentally different therapeutic approaches. While ischemic stroke is commonly treated with thrombolytic and antiplatelet therapy, these interventions are contraindicated in hemorrhagic stroke, in which rapid life-saving measures and intracranial pressure management are the primary therapeutic priorities (Bakri et al., 2026).

Spontaneous intracerebral hemorrhage is primarily caused by rupture of small intracerebral vessels, resulting in acute neurological deficits and symptoms related to increased intracranial pressure, including severe headache, vomiting, impaired consciousness, and focal neurological deficits (Surdji et al., 2025). Chronic hypertension is the most common underlying etiology, contributing to degenerative changes in small cerebral arteries, fibrinoid necrosis, lipohyalinosis, and the formation of Charcot–Bouchard microaneurysms that are prone to rupture during sudden

elevations in blood pressure (Andrian & Wahyuni, 2023; Muhammad & Nabila, 2023). These pathological changes account for the high mortality and poor functional outcomes associated with spontaneous ICH.

Although hemorrhagic stroke predominantly affects older adults, it may also occur in younger individuals. In this population, rupture of an arteriovenous malformation (AVM) represents one of the most frequent causes of spontaneous intracerebral hemorrhage (Magid et al., 2022). AVMs are congenital vascular malformations characterized by abnormal direct connections between arteries and veins without an intervening capillary network, creating high-flow vascular shunts that predispose patients to intracranial hemorrhage (Maurae et al., 2025). Clinical manifestations of ruptured AVMs commonly include sudden severe headache, nausea, vomiting, seizures, impaired consciousness, and focal neurological deficits such as hemiparesis. Hemorrhage may rapidly progress to cerebral edema, elevated intracranial pressure, brain herniation, permanent neurological impairment, or death if not promptly recognized and treated.

Management of spontaneous intracerebral hemorrhage aims to stabilize the patient, control blood pressure, reduce intracranial pressure, prevent secondary brain injury, and determine the need for neurosurgical intervention. Mannitol, an osmotic diuretic, remains one of the most widely used agents for reducing cerebral edema and intracranial pressure in patients with acute brain injury. By increasing plasma osmolality, mannitol facilitates the movement of water from the cerebral parenchyma into the intravascular compartment, thereby reducing brain swelling and improving cerebral perfusion. Nevertheless, evidence regarding its effectiveness in young adults with AVM-related intracerebral hemorrhage remains limited, particularly among patients managed conservatively without surgical intervention.

Herein, we present the case of a 22-year-old man with spontaneous intracerebral hemorrhage secondary to ruptured arteriovenous malformation who was successfully managed with conservative treatment, including intravenous mannitol. Despite extensive intracerebral hemorrhage with intraventricular extension, the patient experienced complete neurological recovery without residual neurological deficits. This case highlights the potential role of conservative intracranial pressure management in carefully selected patients with AVM-related intracerebral hemorrhage and reviews the current evidence regarding the use of mannitol in this clinical setting.

RESULTS AND DISCUSSION

A 22-year-old man presented to the emergency department with a sudden onset of severe headache approximately 15 minutes before admission, accompanied by nausea, vomiting, and decreased consciousness. He had no documented history of hypertension, diabetes mellitus, coagulation disorders, or previous cerebrovascular disease.

Initial non-contrast brain computed tomography (CT) and multislice CT (MSCT) angiography (**Figure 1**) demonstrated a left parieto-occipital intracerebral hemorrhage measuring approximately 5.9 × 4.0 × 5.0 cm, with an estimated hematoma volume of 61.36 mL. The hemorrhage was associated with perifocal cerebral edema and extension into the left lateral ventricle, indicating a severe intracerebral hemorrhage. MSCT angiography further revealed an arteriovenous malformation (AVM) measuring 1.3 × 0.8 × 1.8 cm in the left occipital lobe. The AVM was supplied by branches of the left posterior cerebral artery and drained into the left occipital cortical vein, confirming ruptured AVM as the source of hemorrhage. Additional imaging findings included compression of the left lateral ventricle and mass effect with displacement of adjacent brain structures secondary to the hematoma and cerebral edema, consistent with increased intracranial pressure. No evidence of intracranial aneurysm or significant stenosis of the major cerebral arteries was identified.

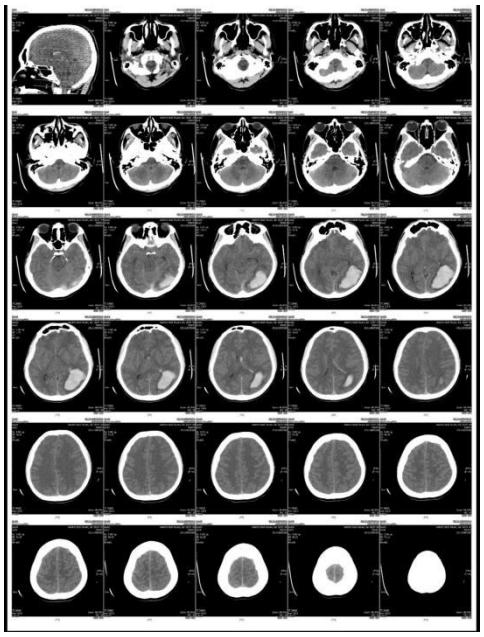

Figure 1. Initial Non-Contrast Brain CT Scan

Laboratory investigations revealed mild hyponatremia with a serum sodium concentration of 132 mmol/L, whereas serum potassium, calcium, chloride, blood urea nitrogen, and creatinine levels were within normal limits, indicating preserved renal function. Coagulation studies showed normal prothrombin time (PT, 14.3 seconds) and activated partial thromboplastin time (APTT, 29.4 seconds). Complete blood count demonstrated a hemoglobin level of 15.1 g/dL, hematocrit of 45.1%, erythrocyte count of $5.15 \times 10^6/\mu L$, and platelet count of $218 \times 10^3/mm^3$, with no evidence of anemia or coagulopathy. However, leukocytosis ($17.89 \times 10^3/mm^3$) with neutrophilia (87.7%), lymphopenia (6.1%), and an elevated neutrophil-to-lymphocyte ratio (NLR) of 14.38 suggested an acute inflammatory or physiological stress response secondary to intracerebral hemorrhage.

Chest radiography demonstrated no cardiopulmonary abnormalities. Cardiac size was within normal limits, pulmonary vascular markings were unremarkable, and no pulmonary infiltrates, pleural effusion, or osseous abnormalities were observed. These findings excluded concomitant cardiopulmonary pathology, supporting the interpretation that the elevated leukocyte count reflected a systemic inflammatory response to acute intracerebral hemorrhage rather than an infectious process.

Given the patient's clinical condition and the absence of immediate indications for emergency neurosurgical intervention, conservative management was initiated. The patient received intravenous mannitol (100–150 mL, four to six times daily) for intracranial pressure reduction, antihypertensive therapy consisting of nifedipine and amlodipine for blood pressure control, tranexamic acid to minimize the risk of further bleeding, and multimodal analgesia including tramadol, paracetamol, ibuprofen, and ketoprofen suppositories. Additional supportive therapy included intravenous ceftriaxone, ranitidine, sucralfate, and 0.9% normal saline infusion. Neurological status, vital signs, and clinical symptoms were closely monitored throughout hospitalization.

The patient's clinical condition progressively improved during hospitalization. His level of consciousness returned to normal with a Glasgow Coma Scale score of E4V5M6, headache intensity decreased to a visual analogue scale (VAS) score of 3, and nausea and vomiting completely resolved. He tolerated oral intake without difficulty and remained neurologically intact throughout the hospital stay. The patient was discharged in stable condition without residual neurological deficits and was scheduled for outpatient follow-up with the neurointerventional service for further evaluation and definitive management of the underlying arteriovenous malformation.

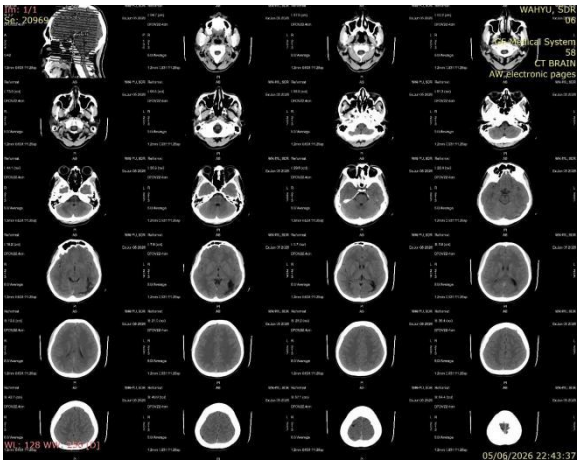

Figure 2. Follow-up Brain CT Scan

Discussion

Intracerebral hemorrhage (ICH) secondary to ruptured arteriovenous malformation (AVM) is a life-threatening neurological emergency that frequently affects younger individuals and is associated with substantial morbidity and mortality. Neurological deterioration following AVM rupture is primarily caused by the combined effects of hematoma expansion, perihematomal edema, and increased intracranial pressure (ICP), all of which contribute to cerebral hypoperfusion and secondary brain injury. Early recognition and timely management aimed at reducing intracranial pressure are therefore essential to optimize neurological outcomes.

In the present case, the patient presented with a large left parieto-occipital intracerebral hemorrhage (approximately 61.36 mL) with intraventricular extension, perifocal edema, and significant mass effect. Despite these radiological findings, the patient remained hemodynamically stable, had preserved renal function, normal coagulation parameters, and no evidence of major systemic complications. These favorable baseline characteristics likely contributed to the successful outcome observed with conservative management.

Mannitol remains one of the most widely used hyperosmolar agents for the treatment of elevated intracranial pressure in patients with acute brain injury. Its therapeutic effect is achieved by increasing plasma osmolality, thereby promoting the movement of water from the cerebral parenchyma into the intravascular compartment, reducing cerebral edema, lowering intracranial pressure, and improving cerebral perfusion. In the present case, intravenous mannitol was administered as part of a comprehensive conservative treatment strategy that also included blood pressure control, analgesia, and supportive care. Progressive neurological improvement was observed throughout hospitalization, with normalization of consciousness (Glasgow Coma Scale score E4V5M6), resolution of headache, nausea, and vomiting, and complete recovery without residual neurological deficits. Follow-up brain CT imaging (**Figure 2**) also demonstrated significant radiological improvement, supporting the favorable clinical course.

The findings of this case are consistent with previous studies demonstrating the potential benefits of mannitol in reducing cerebral edema and controlling intracranial pressure. Husodo et al. (2023) reported that conservative management using mannitol without surgical intervention was associated with clinical improvement in patients with chronic subdural hematoma by reducing progressive cerebral edema and intracranial pressure. Although the underlying pathology differs from AVM-related ICH, both conditions share common pathophysiological mechanisms involving intracranial hypertension and cerebral edema. Similarly, Gunawan (2024) reported that mannitol effectively reduced cerebral edema and intracranial pressure in patients with acute intracerebral hemorrhage, thereby contributing to neurological stabilization.

Current evidence also supports the routine use of mannitol as part of conservative management for hemorrhagic stroke with elevated intracranial pressure. Aisyah et al. (2023) described mannitol as a commonly prescribed osmotic agent, typically administered over 4–7 days with gradual tapering to

minimize cerebral edema and prevent neurological complications. The treatment approach in our patient was consistent with these recommendations and was associated with continuous neurological recovery throughout hospitalization.

Recent literature further supports the role of hyperosmolar therapy in patients with intracerebral hemorrhage. Xiang et al. (2025) reported superior clinical and radiological outcomes among patients receiving mannitol-containing treatment compared with controls. Tian et al. (2022) demonstrated that mannitol administration was associated with reduced perihematomal edema, improved neurological function, and better overall clinical outcomes in patients with intracerebral hemorrhage. Furthermore, Krishnan et al. (2025) identified mannitol as the most frequently used hyperosmolar agent for managing cerebral edema following ICH, while Chen et al. (2025) emphasized that patient selection remains essential because renal function should be carefully evaluated before initiating hyperosmolar therapy.

Although this case demonstrated an excellent neurological outcome, the favorable recovery cannot be attributed solely to mannitol therapy. Several additional factors likely contributed to the patient's recovery, including his young age, preserved physiological reserve, stable hemodynamic status, absence of coagulopathy, normal renal function, meticulous neurological monitoring, and comprehensive supportive management. Therefore, this report should not be interpreted as evidence that conservative therapy is superior to surgical intervention but rather as an illustration that carefully selected patients with AVM-related intracerebral hemorrhage may achieve favorable outcomes with individualized conservative management when immediate neurosurgical intervention is not indicated.

As a single case report, this study has inherent limitations and cannot establish causality or generalize treatment efficacy. Nevertheless, it provides additional clinical evidence supporting the potential role of conservative management, including mannitol, in selected young adults with AVM-related intracerebral hemorrhage. Larger prospective studies are needed to determine the optimal indications, dosing strategies, and long-term outcomes of hyperosmolar therapy in this patient population.

CONCLUSION

This case demonstrates that conservative management incorporating intravenous mannitol may be associated with favorable neurological outcomes in carefully selected young adults with intracerebral hemorrhage secondary to ruptured arteriovenous malformation (AVM). Despite a large parieto-occipital hematoma with intraventricular extension and cerebral edema, the patient achieved complete neurological recovery without residual deficits following comprehensive medical management and close neurological monitoring. The favorable outcome was likely influenced not only by hyperosmolar therapy but also by the patient's young age, preserved organ function, stable hemodynamic status, and individualized supportive care. Although surgical intervention remains the definitive treatment for many patients with ruptured AVM, this case suggests that conservative therapy may represent a reasonable initial management strategy in selected clinically stable patients. Further prospective studies are needed to better define the role of conservative management and the optimal use of mannitol in AVM-related intracerebral hemorrhage.

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