Cerebellar Intracerebral Hemorrhage with Intraventricular Extension: A Case Report in a 62-Year-Old Male with Uncontrolled Hypertension

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ABSTRACT

Intracerebral hemorrhage (ICH) is a subtype of stroke with high morbidity and mortality, where cerebellar location with intraventricular extension (IVH) is a serious condition. Uncontrolled hypertension is a major risk factor that plays a role in the occurrence of ICH. This study is a case report of a 62-year-old man with a history of uncontrolled hypertension who presented with decreased consciousness accompanied by nausea and vomiting. CT scan examination showed left cerebellar hemorrhage measuring ±18.11 cc with extension to the entire ventricular system. The patient was managed conservatively with blood pressure control, intracranial pressure reduction, and supportive therapy. During treatment, the patient experienced improved consciousness, blood pressure stabilization, and neurological function without progressive complications. This case demonstrates that appropriate and comprehensive conservative management can provide good clinical outcomes in patients with cerebellar ICH with IVH extension. This case report emphasizes the importance of early detection, appropriate intervention, and control of risk factors, especially hypertension.

Keyword : Hypertension; Intracerebral hemorrhage; Intraventricular hemorrhage; Cerebellum

Introduction

Intracerebral hemorrhage (ICH) is a severe subtype of stroke and remains one of the leading causes of neurological morbidity and mortality. This subtype occurs within the brain parenchyma, with or without extension into the ventricles[1]. ICH is the second most common type of stroke, accounting for 27.9% of all stroke cases after ischemic stroke (62.4%)[2]. More specifically, cerebellar ICH represents approximately 10% of all ICH cases and 1.5% of all stroke incidences[3]. An epidemiological meta-analysis reported that the global incidence of ICH has not declined over the past 40 years, remaining at 29.9 cases per 100,000 individuals per year, with the Asian population having the highest incidence compared to other population[4]. In Indonesia, data from 1990–2021 revealed that the age-standardized mortality rate reached 112.57 per 100,000 population [5]. Therefore, ICH remains a critical global health issue requiring substantial attention in terms of management.

Uncontrolled hypertension is the primary risk factor, associated with 65% of ICH cases. Hypertensive ICH typically occurs around or at the branching points of small penetrating arteries originating from the anterior, middle, or posterior cerebral arteries, or the basilar artery[2]. Other modifiable risk factors include smoking, alcohol consumption, anticoagulant use, low-density lipoprotein cholesterol reduction, low triglyceride levels, antithrombotic agents, and sympathomimetic drugs. Non-modifiable risk factors comprise advanced age, male sex, cerebral amyloid angiopathy, and Asian ethnicity[6].

Intraventricular hemorrhage (IVH) extension represents one of the major complications and an independent predictor of worse outcomes and neurological deterioration in patients with ICH, particularly spontaneous ICH[7]. IVH extension in ICH cases is associated with increased mortality and poorer functional prognosis[8]. Pathophysiologically, blood entering the ventricular system can trigger inflammatory responses, raise intracranial pressure, and cause secondary damage to surrounding brain tissue, thereby worsening the clinical condition. A study reported that approximately 40% of patients with ICH experience IVH extension, which is significantly correlated with reduced survival rates [9,10]. Therefore, early identification and timely, aggressive management of ICH patients with IVH extension are crucial to improving survival rates and minimizing long-term disability.

Cerebellar ICH with IVH extension presents a distinct clinical challenge due to its often non-specific and variable manifestations, ranging from severe headache, nausea, vomiting, seizures, neurological deterioration, contralateral sensorimotor deficits, to impaired consciousness, which may mimic other neurological conditions [6]. The difficulty in

establishing an early clinical diagnosis without supportive investigations often leads to delays in intervention, consequently increasing the risk of morbidity and mortality. Hence, brain imaging with computed tomography (CT) scan plays a critical role as the initial diagnostic modality, being rapid, accurate, and widely accessible. CT scans not only allow for the identification of the location and size of cerebellar ICH but also determine the presence of IVH extension [10,11].

This case report is presented to highlight the clinical challenges in managing cerebellar ICH with IVH extension in a patient with uncontrolled hypertension. Although rare, this condition carries a high risk of serious complications and mortality if not diagnosed and treated promptly and appropriately. This report aims to contribute to both the literature and clinical practice by emphasizing variations in presentation and management strategies for cerebellar ICH with IVH extension, particularly in patients with uncontrolled hypertension.

Case Illustration

A 62-year-old male was referred to Dr. Soegiri General Hospital on April 26, 2025, with complaints of decreased consciousness accompanied by nausea and five episodes of vomiting. The initial vomiting consisted of food contents, followed by blackish vomitus. Upon arrival at the emergency department of Dr. Soegiri General Hospital, his vital signs showed blood pressure of 160/90 mmHg, pulse rate of 100 beats per minute, SpO_2 98%, and the initial neurological examination revealed a Glasgow Coma Scale (GCS) score of E2V2M3. A few hours later, his level of consciousness deteriorated to delirium and subsequently somnolence, accompanied by an increase in blood pressure to 180/100 mmHg, and further elevation on the following day reaching 191/103 mmHg.

Initial diagnostic workup included a head CT scan, which demonstrated a hyperdense area (67 HU) measuring approximately 3 × 4.3 × 2.7 cm (volume approximately 18.11 cc) in the left cerebellar hemisphere, with intraventricular extension filling both lateral ventricles, the third ventricle, and the fourth ventricle, without evidence of midline shift (**Figure 1**). The radiological conclusion was left cerebellar intracerebral hemorrhage (ICH) with intraventricular hemorrhage (IVH). Hematological results showed hemoglobin 15.9 g/dL, leukocytes 13,260/µL, platelets 235,000/µL, and neutrophil-to-lymphocyte ratio (NLR) 19.44.

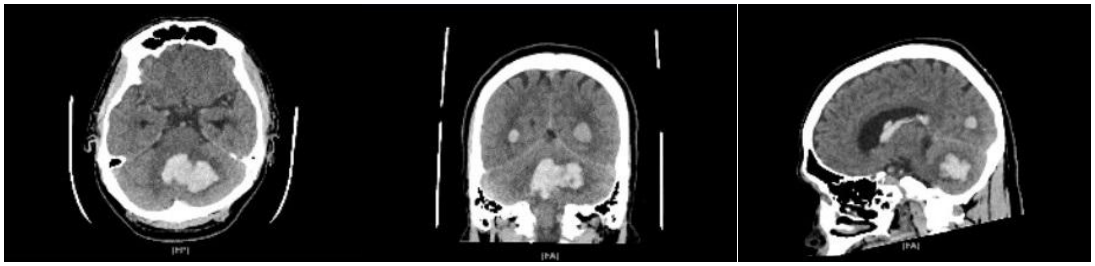

Figure 1. Head CT scan findings at the time of the patient's arrival at the emergency department.

The patient was admitted to the inpatient ward for conservative management. Oxygen supplementation was administered via nasal cannula at 4 L/min. Blood pressure control was achieved using intravenous nicardipine infusion at 5 cc/hour with a target systolic blood pressure of 140–160 mmHg, along with oral amlodipine 10 mg once daily and candesartan 16 mg once daily. Intracranial pressure reduction was managed with an intravenous loading dose of mannitol 200 mL, followed by maintenance therapy with 100 cc administered six times, then gradually tapered according to clinical progression. Neuroprotective therapy included citicoline 250 mg intravenously twice daily, initiated from the beginning of treatment. Supportive therapy consisted of omeprazole 40 mg intravenously twice daily, sucralfate syrup three times daily, acetazolamide 250 mg three times daily, ondansetron 4 mg intravenously twice daily, and diphenhydramine 1 g intravenously three times daily. Intravenous fluids included Ringer's lactate 1000 mL/24 hours and normal saline. The patient was also counseled to follow a low-salt diet.

During hospitalization, the patient underwent close monitoring of vital signs and neurological status. Blood pressure fluctuations with several hypertensive episodes required adjustment of nicardipine dosage before eventually stabilizing within the target range. Mannitol was administered as scheduled in the early phase and gradually reduced as the clinical condition improved. The patient's consciousness showed progressive improvement, evolving from somnolence to increased responsiveness, and ultimately reaching full compos mentis towards the end of hospitalization. Clinical improvement was further evidenced by recovery of consciousness, absence of intracranial pressure elevation, gradual improvement of right extremity muscle strength, reduction of right-hand swelling, and the ability to tolerate oral feeding. Repeat laboratory tests revealed hypokalemia (potassium 3.1 mmol/L), hemoglobin 17.2 g/dL, leukocytes 14,210/µL, platelets 155,000/µL, and NLR 9.39. Potassium correction was carried out with intravenous KCl 50 mEq/24 hours. No further signs of increased intracranial pressure were observed, and antihypertensive as well as

neuroprotective therapies were continued.

On May 7, 2025, the patient was in good general condition, with stable vital signs, oxygen saturation of 97–99% without supplemental oxygen, and was able to communicate and eat well. The patient was deemed fit for discharge with advice to undergo outpatient follow-up for continued monitoring.

Discussion

ICH is a subtype of stroke associated with high morbidity and mortality rates. Cerebellar ICH accounts for only a small proportion of all ICH cases but has serious consequences due to the risk of brainstem compression or hydrocephalus resulting from IVH extension. Uncontrolled hypertension is the dominant risk factor in most cases, as seen in the patient presented in this case report. In this case, the patient was a 62-year-old male with a history of uncontrolled hypertension, which represents the primary risk factor for ICH. Advanced age and male sex also contributed as non-modifiable risk factors, while hypertension remained the most important determinant in triggering cerebellar ICH with IVH extension. The combination of these risk factors explains the patient's susceptibility to the clinical event experienced.

Although uncontrolled hypertension was the dominant risk factor in this patient, it is also important to note that there are several other risk factors that have been known to contribute to ICH. The use of anticoagulation and antiplatelet agents has been associated with an increased risk of ICH and worse early outcomes, and is therefore an important consideration when reviewing patients with ICH [12,13]. Additionally, smoking and tobacco use have also been associated with a higher risk of ICH in some studies [4–16]. Finally, lipid status shows a more complex relationship, where several studies have identified an association between cholesterol level and an increased risk of ICH [17]. These factors, although not present in this case, provide a more comprehensive clinical context and remind physicians to review medication history, smoking status, and lipid profile when assessing risk in patients with ICH.

The patient presented with decreased consciousness accompanied by nausea and vomiting, which are typical manifestations of cerebellar ICH [18]. The initial findings in the emergency department also showed elevated blood pressure with declining consciousness, reflecting the progressive neurological deterioration. Such findings are commonly observed after ICH. This condition is driven by a combination of factors and mechanisms, including increased intracranial pressure, premorbid hypertension, activation of neurovegetative

signaling, and neuroendocrine pathways [19]. A pooled analysis study demonstrated that stable and early systolic blood pressure reduction is safe and associated with favorable outcomes in patients with ICH [20]. Therefore, in this case, immediate blood pressure control was initiated in the emergency department, with a target systolic blood pressure of 140–160 mmHg.

Management in this patient focused on blood pressure control, intracranial pressure reduction, and comprehensive supportive therapy. Blood pressure was managed with intravenous nicardipine infusion titrated dynamically according to fluctuations. Evidence indicates that nicardipine administration after symptom onset provides intensive blood pressure reduction, limits hematoma expansion, and improves functional outcomes [21]. Moreover, another study reported that intravenous nicardipine achieved blood pressure targets more rapidly compared to labetalol and was safer than nitroprusside regarding mortality risk [22]. This strategy aligns with the principle that controlled blood pressure reduction can prevent hematoma expansion without compromising cerebral perfusion. In addition to intravenous antihypertensives, the patient also received oral therapy with a combination of amlodipine and candesartan. A recent phase III randomized clinical trial reported that the amlodipine–candesartan combination showed superior efficacy and good tolerability compared with amlodipine alone, making this combination promising for patients with uncontrolled hypertension [23]. In this case, intravenous nicardipine therapy accompanied by oral antihypertensive combination of amlodipine and candesartan successfully achieved clinical improvement and target blood pressure within a short period.

Intracranial pressure management was also a focus of treatment through intravenous mannitol administration. The American Stroke Association/American Heart Association guidelines recommend mannitol therapy guided by intracranial pressure monitoring, which has been shown to improve prognosis and reduce mortality in patients with ICH or IVH, or ICH with IVH extension accompanied by decreased consciousness. Intracranial pressure–guided mannitol therapy in patients with ICH and impaired consciousness is therefore expected to provide a favorable prognosis [24]. This is consistent with the clinical outcome in this case, where improvement in intracranial adaptive capacity was demonstrated by the absence of further intracranial pressure elevation. These results are supported by an observational study conducted in Indonesia, where tapering mannitol administration with short-term duration resulted in favorable clinical outcomes without adverse effects [25]. Clinical improvement may be explained by the multifactorial mechanisms of mannitol, including increasing intravascular tonicity, inhibiting cerebrospinal fluid (CSF) secretion,

enhancing CSF absorption, reducing CSF volume, lowering free radical levels, attenuating ischemia–reperfusion injury, inhibiting apoptosis, and improving cerebral perfusion pressure [26].

Supportive therapy was also provided, as it plays a crucial role in maintaining systemic stability and facilitating recovery. Intravenous citicoline was administered as a neuroprotective agent to restore neuronal cell membrane function. Citicoline acts as a precursor in phosphatidylcholine synthesis, thereby aiding in the repair and stabilization of damaged neuronal membranes following ICH. Furthermore, citicoline enhances phospholipid metabolism, reduces accumulation of neurotoxic free fatty acids, and decreases oxidative stress. In addition, citicoline stimulates acetylcholine and dopamine synthesis, supporting neurotransmitter function [27]. Clinically, evidence indicates that citicoline administration is associated with improvement in the National Institute of Health Stroke Scale (NIHSS) score in stroke patients [28]. These therapeutic effects underline its potential as a neuroprotective agent in ICH by preserving cell membrane integrity, mitigating secondary tissue injury, and enhancing neurological recovery.

In this case, recurrent vomiting culminating in blackish vomitus was observed. This finding is consistent with recent evidence showing that gastrointestinal bleeding frequently occurs in patients with ICH, often presenting as hematemesis or brownish/black vomiting [29]. This condition is thought to be associated with heightened physiological stress, use of certain medications, and hemodynamic disturbances accompanying acute neurological disease. Therefore, gastrointestinal protection therapy with omeprazole and sucralfate was administered to prevent gastrointestinal bleeding complications [30]. To manage nausea and vomiting, ondansetron was given, which effectively suppresses the vomiting reflex in patients with neurological and systemic conditions. Additionally, ondansetron modulates neurotransmitter imbalance, making it effective for controlling emesis in stroke patients [31]. The patient also received acetazolamide, which plays a role in regulating cerebrospinal fluid dynamics, thereby contributing to intracranial pressure control and potentially preventing symptom progression in ICH with IVH extension [32]. This pharmacotherapeutic approach highlights the importance of multidisciplinary supportive management, not only focused on neurological stabilization but also on preventing systemic complications that may worsen prognosis in patients with ICH and IVH extension.

This case emphasizes that cerebellar ICH with IVH extension in patients with uncontrolled hypertension constitutes a neurological emergency requiring aggressive, multidisciplinary management from the early phase. The combined strategy of strict blood

pressure control, intracranial pressure management, and comprehensive supportive and neuroprotective therapy proved effective in improving clinical outcomes in this patient. Appropriate conservative management may be an option in patients with stable conditions without progressive intracranial pressure elevation. From a clinical perspective, this case report implies that self-intervention, risk factor control, and comprehensive management not only enhance recovery potential but also reduce morbidity and mortality in patients with ICH and IVH extension.

Conclusion

This case report demonstrates that cerebellar ICH with IVH extension in a patient with uncontrolled hypertension represents a serious condition with a high risk of mortality, yet it can be effectively managed through comprehensive treatment. A multidisciplinary approach consisting of strict blood pressure control, intracranial pressure management, and supportive as well as neuroprotective therapy proved successful in improving the patient's clinical condition and outcomes. This case highlights the importance of early detection, timely intervention, and rigorous risk factor control, particularly hypertension, as key strategies to reduce morbidity and mortality in patients with ICH.

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