



# NONTRAUMATIC SPONTANEOUS BILATERAL INTRACEREBRAL HEMORRHAGE: A RARE CASE SERIAL

Rosalina Pradana Ayu\*, Raden Andi Ario Tedjo, Siti Roisya Aga Maydiana

\*Correspondence: [rosalina.pradana@gmail.com](mailto:rosalina.pradana@gmail.com)

Department of Neurology, Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Indonesia

## Article History:

Received: July 19, 2024  
 Accepted: August 29, 2024  
 Published: July 1, 2025

## Cite this as:

Ayu RP, Tedjo RAA, Maydiana SRA. Nontraumatic Spontaneous Bilateral Intracerebral Hemorrhage: A Rare Case Serial. *Magna Neurologica*. 3(2) July 2025: 108-112. 10.20961/magnaneurologica.v3i2.1731

## ABSTRACT

**Background:** Nontraumatic spontaneous bilateral intracerebral bleeding is infrequent, with only 30-40 cases reported worldwide. Hypertension is the leading cause due to the formation of microaneurysms at the lenticulostriate arteries. Treatment follows current intracerebral hemorrhage guidelines, focusing on supportive care and early blood pressure management. This case report aims to further understand nontraumatic spontaneous bilateral intracerebral hemorrhage.

**Case:** We report two cases of nontraumatic bilateral spontaneous intracerebral hemorrhage with similar etiologies. The first case involved a 34-year-old male with weakness in the right extremities, slurred speech, and high blood pressure (213/138 mmHg). The second case involved a 65-year-old woman with weakness in the left extremities, slurred speech, and high blood pressure (175/95 mmHg). Both patients had bilateral hemorrhages in different areas of the brain. They were treated according to management guidelines for intracerebral hemorrhage and showed improvement.

**Discussion:** Nontraumatic spontaneous bilateral intracerebral bleeding is often caused by hypertension. CT angiography is a vital test for identifying vascular abnormalities. Treatment involves reducing risk factors for complications and controlling blood pressure. Typically, management of this condition is conservative, and unfavorable outcomes are common. However, these patients showed improvement.

**Conclusion:** Conservative management, supportive care, and early blood pressure management for nontraumatic spontaneous bilateral intracerebral hemorrhage patients can cause improvement in the patient's condition.

**Keywords:** bilateral intracerebral, hemorrhagic stroke, uncontrolled hypertension



This is an open access article distributed under the terms of the Creative Commons Attribution-4.0 International License

## Introduction

Intracerebral hemorrhage (ICH) refers to the rapid development of neurological dysfunction caused by blood collection in the brain or ventricular system.<sup>1</sup> It accounts for 10-20% of stroke etiologies annually.<sup>2</sup> The classification of ICH is based on its anatomical location, such as deep subcortical ICH or lobar hemorrhage.<sup>1</sup> Meanwhile, non-traumatic bilateral spontaneous intracerebral hemorrhage is a scarce type of ICH, occurring in only 0.3%-0.8% of cases worldwide.<sup>3,4</sup> Spontaneous bilateral intracerebral

hemorrhage was also poorly studied, with only 30-40 reported cases.<sup>20</sup>

Basal ganglia hemorrhages can be classified into traumatic and non-traumatic categories, with non-traumatic cases having various underlying causes. These causes can be categorized using two classification systems:

1. H-ATOMIC: This acronym represents Hypertension, Amyloid Angiopathy, Tumor, Oral anticoagulant, Malformation, Infrequent, Cryptogenic, and Combination.
2. SMASH-U: This classification includes Structure

vascular lesion, Medication, Amyloid angiopathy, Systemic disease, Hypertension, and undetermined causes.<sup>1,3</sup>

Hypertension is the most common underlying cause for basal ganglia hemorrhage, accounting for 50% of cases, followed by intoxication and metabolic causes.<sup>5</sup> Hypertension damages blood vessels, leading to microaneurysms, lipohyalinosis, and arterial dissection. Thus, the risk of microaneurysm rupture is increased abruptly. Spontaneous bilateral intracerebral hemorrhage induced by hypertension typically occurs in individuals with a long history of poorly treated hypertension.<sup>5</sup> Bilateral intracerebral hemorrhage occurs when a microaneurysm in the lenticulostriate artery ruptures and the thalamic artery perforates simultaneously.<sup>5</sup> It can also occur due to the simultaneous rupture of microaneurysms in the lenticulostriate arteries. Another possible explanation is that the initial bleeding triggers hemodynamic conditions that lead to the rupture of a second microaneurysm on the opposite side.<sup>6</sup> Computed tomography (CT) scanning is reliable for diagnosing cerebral and intraventricular hemorrhage. CT Angiography can provide additional information on potential subsequent bleeding, aneurysms, and arteriovenous malformations.<sup>7</sup> Treatment for intracerebral hemorrhage is primarily conservative, focusing on supportive care and managing hypertension.<sup>8</sup>

## Case Report

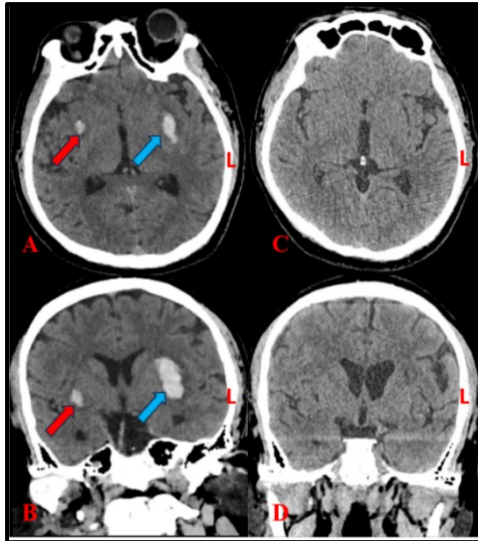
This case series includes two patients with non-traumatic bilateral spontaneous intracerebral hemorrhage. Here are the key details:

1. Patient 1: A 34-year-old man presented with sudden right limb weakness, dysarthria, and high blood pressure. Imaging revealed blood-density lesions in the left and right lentiform nuclei (Figure 1). The patient received conservative management for six days and showed improvement, leading to discharge. Follow-up imaging showed complete absorption of the blood.
2. Patient 2: A 65-year-old woman arrived at the hospital with sudden left limb weakness, dysarthria, and high blood pressure. Imaging revealed multiple blood-density lesions extending from the posterior crus of the right internal capsule to the right thalamus and in the left thalamus (Figure 2). The patient received conservative management for six days and showed improvement, leading to discharge. Follow-up imaging showed complete absorption of the blood.

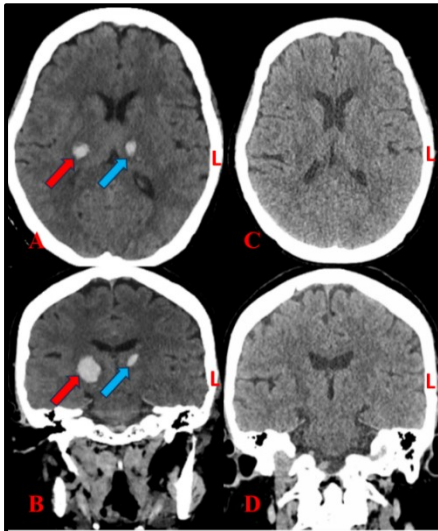
Both patients had similar presentations and positive outcomes (Table 1).

**Table 1.** Bilateral Intracerebral Hemorrhage Case Series

Case	I	II
<b>Age, Gender</b>	Mr. R, 34 Years old, Male	Mrs. S, 65 Years old, Female
<b>Onset</b>	3 hours before admission	2 hours before admission
<b>GCS</b>	E4V5M6	E4V5M6
<b>Nutritional Status</b>	Standard (BMI 25 kg/m <sup>2</sup> )	Overweight (BMI 25.4 kg/m <sup>2</sup> )
<b>Blood Pressure</b>	213/138 mmHg (MAP 163 mmHg)	175/95 mmHg (MAP 122 mmHg)
<b>Speech</b>	Dysarthria	Dysarthria
<b>Cranial Nerve</b>	Paresis N.VII and XII dextra, UMN	Paresis N.VII and XII sinistra, UMN
<b>Motor System</b>	Hemiparesis dextra 444/555 444/555	Hemiparesis sinistra 555/333 555/333
<b>Pathologic Reflex</b>	Hoffman Tromner (+/-) Babinski Reflex (+/-)	Hoffman Tromner (-/+) Babinski Reflex (-/+)
<b>Scoring during Admission</b>		
<b>ICH</b>	0	0
<b>NIHSS</b>	6	10
<b>SICH</b>	2	2
<b>FUNC</b>	10	10
<b>mRS</b>	2	3
<b>Total Cholesterol</b>	178 mg/dl	233 mg/dl
<b>LDL</b>	132 mg/dl	142 mg/dl
<b>ECG</b>	Normal sinus rhythm 90 bpm	Normal sinus rhythm 75 bpm
<b>Chest X-ray</b>	Normal	Cardiomegaly
<b>Non-contrast Head CT during Admission</b>	ICH in bilateral lentiform nuclei	ICH in bilateral thalamus
<b>CT Angiography during Treatment (Figure 3)</b>	ICH in bilateral lentiform nuclei; no aneurysms, AMVs, or vascular malformations	ICH in bilateral thalamus; no aneurysms, AMVs, or vascular malformations
<b>Hemorrhage Volume</b>	Right: 0,5 cc Left: 10,5 cc	Right: 4 cc Left: 0.6 cc
<b>Treatment</b>	Conservative	Conservative
<b>Treatment Duration</b>	6 days	6 days
<b>mRS 1 month</b>	1	1
<b>mRS 3 months</b>	1	1
<b>mRS 6 months</b>	1	1
<b>Non-contrast Head CT-6 6-month evaluation</b>	Completely Absorbed	Completely Absorbed



**Figure 1.** Non-contrast head CT of Mr. R: (A) axial and (B) coronal views show bilateral hyperdense lesions in the right (red arrow) and left (blue arrow) lentiform nuclei. Complete resorption in 6 months (C, D)



**Figure 2.** Non-contrast head CT of Mrs. S: (A) axial and (B) coronal views show bilateral hyperdense lesions in the right (red arrow) and left (blue arrow) thalami. Complete resorption in 6 months (C, D)



**Figure 3.** (A) Mr. R's and (B) Mrs. S CT angiography scan shows that there were no aneurysms, AVMs, or vascular malformations found

## Discussion

Non-traumatic intracerebral hemorrhage (ICH) is a type of stroke where blood leaks into the brain tissue without any head trauma. It is categorized as a hemorrhagic stroke, different from ischemic stroke, where a cerebral artery is blocked.<sup>9</sup> This condition affects around 2 million people worldwide each year.<sup>9</sup> The most common type of non-traumatic ICH is the non-lobar type, which is caused by prolonged high blood pressure leading to the damage of small intracerebral arteries, thalamus, pons, and microaneurysms of the cerebellum. It is often associated with lipo-hyalinosis.<sup>3,9,10</sup>

Simultaneous bilateral non-traumatic basal ganglia hemorrhage is a sporadic form of intracerebral hemorrhage (ICH), primarily observed in Asians, males, and older people.<sup>11</sup> Risk factors for this condition include chronic kidney failure, cerebral microhemorrhage, and cerebral amyloid angiopathy, with untreated hypertension being the leading cause.<sup>3,12</sup> Two critical mechanisms contribute to intracerebral hemorrhage. The first is the rupture of small penetrating arteries due to chronic hypertension and aging, making them more susceptible to rupture.<sup>13</sup> The second mechanism involves an acute increase in blood pressure, leading to the rupture of normal arteries and capillaries. Interestingly, many patients with intracerebral hemorrhage do not have a history of hypertension, and signs of chronic hypertension may be absent. Acute blood pressure and blood flow increases can trigger the rupture of previously unaffected arterioles and capillaries. The risk of rupture tends to be higher with more drastic increases in blood pressure.<sup>13,14</sup>

Bilateral intracerebral bleeding is usually caused by the rupture of a microaneurysm, which occurs at the location of the lenticulostriate artery and simultaneously perforates the thalamic artery.<sup>3,15</sup> Other known causes of multiple intracerebral hemorrhages are hemorrhagic diathesis, cerebral amyloid angiopathy, vasculitis, sinus thrombosis, neoplasms, aneurysms, arteriovenous malformations, angiomas, and drug abuse.<sup>6,16</sup>

In this case, two patients experienced non-traumatic bilateral spontaneous intracerebral hemorrhage. The exact mechanism for this condition is still uncertain, but there are two main hypotheses. One suggests that bilateral micro-aneurysms rupture or arteries with hyaline degeneration perforate simultaneously. The other proposes that initial bleeding occurs unilaterally, with patients later developing bilateral symptoms due to structural or hemodynamic changes, leading to secondary bleeding.<sup>4,9</sup>

Both patients had a history of hypertension and advanced cerebrovascular degeneration, which increased their risk for secondary bleeding. However, there were no other causative factors, such as coagulopathic

dyslipidemia or long-term use of antiplatelets or anticoagulants. CT angiography did not reveal other possible causes, such as arteriovenous malformation or aneurysms.<sup>17</sup>

In this case, conservative management is implemented to control blood pressure and minimize the risk of complications. The INTERACT-2 and ATACH-2 clinical trials have demonstrated that aggressively reducing systolic blood pressure within 4.5 hours after onset can prevent hematoma expansion and improve clinical outcomes. Lowering systolic blood pressure to < 140 mmHg reduces the risk of neurological damage, while maintaining it below < 160 mmHg is associated with a better long-term prognosis.<sup>15,18</sup> After a CT scan 6 months after onset, it was observed that the hematomas in both patients had been utterly absorbed. On average, hematomas tend to resorb at 1.02ml/day.<sup>19</sup>

Complications of non-traumatic bilateral spontaneous intracerebral hemorrhage include cerebral edema, aspiration pneumonia, quadriparesis, hemiparesis, and recurrent stroke. Supportive management aims to reduce risk factors, primarily by controlling blood pressure.<sup>20</sup> Keeping blood pressure below 140/90 improves functional outcomes without reducing mortality or severe disability. However, a more aggressive target of below 120 mmHg is not recommended, especially in acute conditions, as it can cause other organ disorders like heart and kidney insufficiency.<sup>3</sup>

## Conclusion

Non-traumatic bilateral intracerebral spontaneous hemorrhage is a rare condition commonly caused by uncontrolled hypertension and vascular anomalies. The diagnosis is typically made using a plain CT scan of the head to differentiate between infarction and hemorrhage. Further vascular studies, such as CTA, are often performed to rule out vascular anomalies, although more minor anomalies may not be detected. In some cases, diagnostic DSA is recommended for a more thorough examination.

The management of bilateral intracerebral hemorrhage depends on the patient's clinical course and imaging results. In these two cases, conservative management was chosen due to the patient's stability, absence of increased intracranial pressure, and small hemorrhage volume. Both patients showed good clinical outcomes with complete blood absorption at the 6-month follow-up, as indicated by a mRS score 1.

## References

1. Alhashim A, Hadhiah K, Al-dandan H, Aljaman M, Alabdali M. Spontaneous Simultaneous Bilateral Basal Ganglia Hemorrhage (SSBBGH): Systematic Review and Data Analysis on Epidemiology, Clinical Feature, Location of Bleeding, Etiology, Therapeutic Intervention and Outcome; 2022. p. 267–76. DOI: 10.2147/VHRM.S349912
2. Ismail R, George DD, Kohli GS, Khan MW, Wang HZ, Mattingly TK. A Case of spontaneous basal ganglia hemorrhage with contralateral extension utilizing the canal of Gratiolet. *BMC Neurol*; 2023. p. 1–5. DOI: 10.1186/s12883-023-03232-4
3. Kahar LA, Ikhsani R, Sari WM. Intensive Management of Spontaneous Basal Ganglia Hemorrhage: A Case Report. *Biosci Med J Biomed Transl Res*; 2023. p. 3587–91. DOI: 10.37275/bsm.v7i9.862
4. Wu TY, Yassi N, Shah DG, Ma M, Sharma G, Putaala J, et al. Simultaneous Multiple Intracerebral Hemorrhages (SMICH). *Stroke*; 2017. p. 581–6. DOI: 10.1161/STROKEAHA.116.015186
5. Mohamed Sheikh Hassan, Ali AM, Mohamed Farah Osman, Ahmed A. Spontaneous Bilateral Basal Ganglia Hemorrhage Due to Severe Hypertension; 2022. p. 473–7. DOI: 10.2147/VHRM.S362059
6. Arumugam A, Nagalingam H, Khong TK. A traumatic spontaneous bilateral basal ganglia hemorrhage in a young adult: A case report. *Indonesian Journal of Neurosurgery*; 2022. 5(2):78–81. DOI: 10.15562/ijn.v5i2.178
7. Magid-Bernstein J, Girard R, Polster S, Srinath A, Romanos S, Awad IA, et al. Cerebral Hemorrhage: Pathophysiology, Treatment, and Future Directions. *Indonesian Journal of Neurosurgery*; 2022. p. 1204–29. DOI: 10.1161/CIRCRESAHA.121.319949
8. Watanabe G, Conching A, Ogasawara C, Chavda V, Bin O, Ali A, et al. Bilateral basal ganglia hemorrhage: A systematic etiologies, management strategies, and clinical outcomes review. *Neurosurg Rev*; 2023. DOI: 10.1007/s10143-023-02044-x
9. Park C, Charalambous LT, Yang Z, Adil SM, Hodges SE, Lee HJ, et al. Inpatient mortality and healthcare resource utilization of nontraumatic intracerebral hemorrhage complications in the US. *Stroke*; 2021. 135(10):1081–90. DOI: 10.3171/2020.8.JNS201839
10. Shen J, Guo F, Yang P, Xu F. Influence of hypertension classification on hypertensive intracerebral hemorrhage location. *J Clin Hypertens (Greenwich)*; 2021. 9:1992–9. DOI: 10.1111/jch.14367
11. Tatlisumak T, Cucchiara B, Kuroda S, Kasner SE, Putaala J. Nontraumatic intracerebral hemorrhage in young adults. *Nat Publ Gr*; 2018. 14(4):237–50. DOI: 10.1038/nrneurol.2018.17
12. Lu P, Cui L, Cao Z, Gu H, et al. Association of sex and age with in-hospital mortality and complications of patients with intracerebral hemorrhage: A study from the Chinese Stroke Center Alliance. *Stroke and Vascular Neurology*; 2023. 6:1–10. DOI: 10.1002/brb3.2846

13. Aiyagari V, Gorelick PB. Hypertension and stroke pathophysiology and management. *Hypertension*; 2016. 68(3):523-530. DOI:10.1007/978-3-319-29152-9
14. Sallinen H, Putaala J, Strbian D. Triggering factors in non-traumatic intracerebral hemorrhage. *Journal of Stroke and Cerebrovascular Diseases*; 2020. 29(8):1–7. DOI: 10.1016/j.jstrokecerebrovasdis.2020.104921
15. Kirshner H, Schrag M. Management of Intracerebral Hemorrhage : Update and Future Therapies. *Curr Neurol Neurosci Rep*; 2021. p. 1–5. DOI: 10.1007/s11910-021-01144-9
16. Elmegiri M, Koivunen R jaakko, Tatlisumak T, Putaala J. MRI Characterization of Non-traumatic Intracerebral Hemorrhage in Young Adults. *Front Neurol*; 2020. 11:558680. DOI: 10.3389/fneur.2020.558680
17. Sani AF, Putri SA, Usman FS. *Konsensus Nasional Neurointervensi*. Surabaya: Universitas Airlangga Press; 2020. p. 129.
18. Hanley DF, Hsu CY, Martin RL, Ph D, Moy CS, Ph D, et al. Intensive Blood-Pressure Lowering in Patients with Acute Cerebral Hemorrhage. *The New England Journal of Medicine*; 2016. p. 1033–43. DOI: 10.1056/NEJMoal603460
19. Zhang T, Wang X, Ma L. Peripheral monocyte count is associated with hematoma clearance after intracerebral hemorrhage : a multicenter retrospective study. *Research Square*; 2022. DOI: 10.21203/rs.3.rs-2317975/v1
20. Shaheed TA, Glover N, Alboiny S. Nontraumatic Spontaneous Bilateral Basal Ganglia Hemorrhage : A Rare Case Report. *Case Reports in Neurology*; 2020. 12(11):10–3. DOI: 10.7759/cureus.11299