

Severe Hypertension in Young Adults with Intracerebral Hemorrhage and Target-Organ Damage: A Two-Case Series from Timor-Leste

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ABSTRACT

Background

Hypertension is a major modifiable risk factor for cardiovascular disease, stroke, chronic kidney disease, and premature death. Although hypertension is more common with increasing age, it is increasingly recognized in young adults. Severe hypertension in this age group is particularly concerning because it may reflect previously unrecognized hypertension or secondary hypertension and can result in premature target-organ damage. We report two young men who presented with severe hypertension complicated by intracerebral hemorrhage and neurological disability.

Case Presentation

Case 1: A 24-year-old man with no previously documented medical illness presented after four days of headache, vomiting, and subsequent loss of consciousness. He was a heavy smoker, unemployed, reported poor sleep, and reported no regular exercise. His mother had poorly controlled hypertension. His admission blood pressure was 218/147 mmHg. He had a Glasgow Coma Scale (GCS) score of 12/15, neck stiffness, bilateral proptosis, and right-sided hemiplegia. Investigations showed marked neutrophilic leukocytosis, severe renal dysfunction, hyperuricemia, and elevated total cholesterol. Chest radiography showed cardiomegaly and bilateral basal opacities, electrocardiography showed left ventricular hypertrophy, and brain computed tomography demonstrated a left temporo-occipital intraparenchymal hemorrhage with compression of the ipsilateral ventricle. Echocardiography demonstrated hypertensive heart disease. He received antihypertensive therapy, antibiotics, diuretics, nitrates, and supportive care. His condition progressively deteriorated, and he died after 18 days of hospitalization.

Case 2: A 25-year-old man presented with headache, vomiting, acute left-sided weakness, and facial palsy, with a blood pressure of 192/132 mmHg. He was a chronic smoker, reported heavy alcohol consumption, was unemployed, reported poor sleep, and reported no regular exercise. There was no reported family

history of hypertension. His GCS score was 14/15. Brain magnetic resonance imaging demonstrated a right-sided intraparenchymal hemorrhage. Renal function and thyroid function tests were normal. Electrocardiography showed mild left ventricular enlargement and left axis deviation, while echocardiography demonstrated mild hypertensive heart disease. Abdominal ultrasonography was unremarkable. He was treated with amlodipine 10 mg once daily, enalapril 10 mg once daily, and hydrochlorothiazide 25 mg once daily. After seven days, his blood pressure decreased to 135/94 mmHg, although substantial neurological deficits persisted. He was discharged after 14 days with continued antihypertensive treatment and physiotherapy. At one-month outpatient follow-up, his blood pressure was 120/70 mmHg, and left-sided muscle strength had improved with regular rehabilitation.

Conclusion

Severe hypertension in young adults may present with catastrophic cerebrovascular complications and evidence of early cardiovascular or renal target-organ damage. Early detection, assessment for secondary causes, appropriate blood pressure management, cardiovascular risk-factor modification, rehabilitation, and long-term follow-up are essential. These cases also illustrate the challenges of managing severe hypertension and its complications in a resource-limited setting.

Keywords: hypertension; young adults; intracerebral hemorrhage; hypertensive heart disease; stroke; target-organ damage; smoking; alcohol; Timor-Leste; case series

INTRODUCTION

Hypertension is a major global health problem and one of the most important modifiable risk factors for cardiovascular disease, stroke, chronic kidney disease, and premature death. The World Health Organization (WHO) estimated that approximately 1.4 billion adults aged 30–79 years had hypertension in 2024, with two-thirds living in low- and middle-income countries. A substantial proportion remain unaware of their condition or do not have adequate blood pressure control [1].

Although hypertension becomes more prevalent with increasing age, it is increasingly recognized in young adults. The 2024 European Society of Cardiology (ESC) Guidelines address young adults as those aged 18–40 years and emphasize cardiovascular risk assessment, lifestyle intervention, and appropriate evaluation of hypertension in this age group [2].

Young-onset hypertension deserves particular attention because secondary hypertension is relatively more common in younger patients. Potential causes include renal parenchymal or renovascular disease, primary aldosteronism, endocrine disorders, medication- or substance-related hypertension, and obstructive sleep apnoea [2].

Severe uncontrolled hypertension may cause acute target-organ injury, including intracerebral hemorrhage, acute kidney injury, acute heart failure, and hypertensive cardiac remodeling [2–4]. Hypertension is also one of the most important modifiable risk factors for intracerebral hemorrhage [3].

We report two young men aged 24 and 25 years who presented with severe hypertension complicated by intracerebral hemorrhage and significant neurological deficits. The cases illustrate the potential severity of hypertension in young adults and the importance of early detection, assessment for secondary causes, and comprehensive long-term management.

Case Presentations

Case 1

A 24-year-old single man with no previously documented significant medical history was referred from Liquiçá Community Health Centre to the Emergency Department with a four-day history of headache. He had one episode of vomiting at home followed by loss of consciousness. He was a heavy smoker,

unemployed, reported poor sleep, and reported no regular exercise. His mother reportedly had poorly controlled hypertension.

On arrival, his blood pressure was 218/147 mmHg. He was drowsy and agitated, with a GCS score of 12/15 (E3, V4, M5). Neurological examination demonstrated neck stiffness and right-sided hemiplegia. Bilateral proptosis was also noted. Respiratory examination revealed transmitted breath sounds and bilateral basal crackles.

Laboratory investigations showed marked leukocytosis, with a white blood cell count of $30.7 \times 10^9/L$ and neutrophils of 96%. Haemoglobin and serum potassium were reportedly within the normal range. Urea was markedly elevated at 41.30 mmol/L, serum creatinine was 1238 $\mu\text{mol/L}$, serum uric acid was 786 $\mu\text{mol/L}$, and total cholesterol was 5.7 mmol/L.

Chest radiography demonstrated cardiomegaly with bilateral basal pulmonary opacities. Electrocardiography demonstrated left ventricular hypertrophy.

Computed tomography of the brain demonstrated a hyperdense intraparenchymal lesion in the left temporo-occipital region with compression of the ipsilateral ventricle, consistent with intracerebral hemorrhage.

Echocardiography demonstrated findings reported as hypertensive heart disease.

The patient was diagnosed with severe uncontrolled hypertension complicated by intracerebral hemorrhage, severe renal dysfunction, and suspected pulmonary infection/sepsis. He was managed with antihypertensive medications, antibiotics, diuretics, nitrates, and supportive treatment.

Despite treatment, his clinical condition progressively deteriorated. Haemodialysis was considered but was not undertaken because of the poor prognosis and lack of agreement from relatives. His condition continued to worsen, and he died after 18 days of hospitalization.

Case 2

A 25-year-old man presented with headache, vomiting, acute left-sided weakness, and left facial palsy. His admission blood pressure was 192/132 mmHg. He was a chronic smoker and reported heavy alcohol consumption. He was unemployed, reported poor sleep, and reported no regular exercise. There was no reported family history of hypertension.

Neurological examination demonstrated a GCS score of 14/15 (E4, V5, M5), left facial palsy, marked left-sided weakness, and an upgoing plantar response on the left. Muscle power was 1/5 in the left upper limb and 2/5 in the left lower limb; power on the right side was 5/5. The remaining systemic examination was unremarkable, and fundoscopy was normal.

Magnetic resonance imaging of the brain demonstrated a right-sided intraparenchymal hemorrhage, corresponding clinically to the patient's left-sided neurological deficits.

Routine laboratory investigations were reportedly within normal limits, including renal function. Thyroid function tests were normal. Electrocardiography demonstrated mild left ventricular enlargement and left axis deviation. Echocardiography demonstrated mild hypertensive heart disease. Abdominal ultrasonography did not demonstrate significant abnormalities.

The patient was treated with amlodipine 10 mg orally once daily, enalapril 10 mg orally once daily, and hydrochlorothiazide 25 mg orally once daily.

Following seven days of treatment, his blood pressure decreased to 135/94 mmHg. However, significant neurological impairment persisted, with muscle power remaining 1/5 in the left upper limb and 2/5 in the left lower limb, while the right side remained 5/5.

The patient was discharged after 14 days of hospitalization with continued antihypertensive treatment and regular physiotherapy. At one-month outpatient follow-up, his blood pressure was 120/70 mmHg, and his left-sided muscle strength had improved after regular rehabilitation sessions.

Comparison of the Two Cases

	Case 1	Case 2
Characteristic	24 years	25 years
Sex	Male	Male
Previous diagnosis of hypertension	Not documented	Not documented
Family history	Mother with poorly controlled hypertension	No reported family history
Smoking	Heavy smoker	Chronic smoker
Alcohol consumption	Not reported	Heavy alcohol consumption
Employment status	Unemployed	Unemployed
Sleep pattern	Poor sleep	Poor sleep
Physical activity	No regular exercise	No regular exercise
Admission blood pressure	218/147 mmHg	192/132 mmHg
Main presentation	Headache, vomiting, loss of consciousness, and right-sided hemiplegia	Headache, vomiting, acute left-sided weakness, and left facial palsy
Glasgow Coma Scale	12/15 (E3, V4, M5)	14/15 (E4, V5, M5)
Brain imaging	Left temporo-occipital intracerebral hemorrhage with compression of the ipsilateral ventricle	Right-sided intraparenchymal intracerebral hemorrhage
Renal involvement	Marked renal dysfunction (urea 41.30 mmol/L; creatinine 1238 μ mol/L)	Renal function within normal limits
Other laboratory findings	WBC 30.7×10^9 /L; neutrophils 96%; uric acid 786 μ mol/L; total cholesterol 5.7 mmol/L	Routine laboratory investigations within normal limits
ECG	Left ventricular hypertrophy	Mild left ventricular enlargement and left axis deviation
Echocardiography	Hypertensive heart disease	Mild hypertensive heart disease
Chest X-ray	Cardiomegaly with bilateral basal pulmonary opacities	No significant abnormality reported
Other investigations	—	Thyroid function tests normal; abdominal ultrasonography unremarkable
Treatment	Antihypertensives, antibiotics, diuretics, nitrates, and supportive treatment	Amlodipine 10 mg once daily + enalapril 10 mg once daily + hydrochlorothiazide 25 mg once daily

Blood pressure response	No documented sustained improvement	135/94 mmHg after 7 days
Neurological outcome during hospitalization	Progressive deterioration	Persistent left-sided neurological deficit
Hospital outcome	Died after 18 days	Discharged after 14 days
Outpatient follow-up	Not applicable because the patient died during hospitalization	At 1 month: BP 120/70 mmHg with improved left-sided muscle strength after rehabilitation
Rehabilitation	Not applicable	Regular physiotherapy/rehabilitation continued after discharge

DISCUSSION

The two cases demonstrate the potential severity of hypertension in young adults. Both patients were younger than 26 years and presented with severe hypertension, intracerebral hemorrhage, and evidence of hypertensive cardiac remodeling. Their presentation is consistent with the concern that severe hypertension in young adults may already be associated with clinically important target-organ damage [2,4].

The admission blood pressures of 218/147 mmHg and 192/132 mmHg were markedly elevated. In a patient with severe hypertension and acute neurological symptoms, hypertensive emergency should be considered when there is evidence of acute target-organ injury. The clinical distinction is based not solely on the blood pressure value but on the presence of acute organ damage [2].

Cerebrovascular Complications

The most clinically important complication in both cases was intracerebral hemorrhage. Case 1 demonstrated a left temporo-occipital hyperdense lesion on CT with ventricular compression, whereas Case 2 demonstrated a right-sided intraparenchymal hemorrhage on MRI.

Hypertension has a strong causal association with intracerebral hemorrhage and is a major modifiable risk factor [3]. The 2022 American Heart Association/American Stroke Association (AHA/ASA) guideline emphasizes rapid diagnosis, appropriate acute blood pressure management, and prevention of recurrent ICH through long-term risk-factor control [3].

For adults with spontaneous ICH presenting with systolic blood pressure between 150 and 220 mmHg and without a contraindication to acute blood pressure lowering, the AHA/ASA guideline states that acute lowering toward a target of 140 mmHg, with maintenance in the range of 130–150 mmHg, is safe and may be reasonable in selected patients. Excessive lowering below 130 mmHg may be harmful [3]. Case 2 presented within this systolic blood pressure range. However, the exact timing, agents, and magnitude of acute blood pressure reduction in both patients could not be completely reconstructed from the available records; therefore, treatment in these cases should not be interpreted as adherence or non-adherence to the guideline.

Hypertensive Cardiac Target-Organ Damage

Both patients demonstrated evidence of cardiac involvement. Case 1 had cardiomegaly on chest radiography, left ventricular hypertrophy on ECG, and echocardiographic findings reported as hypertensive heart disease. Case 2 had mild left ventricular enlargement on ECG and mild hypertensive heart disease on echocardiography.

Left ventricular hypertrophy and other structural cardiac changes are recognized markers of hypertension-mediated organ damage and are relevant to cardiovascular risk assessment [2]. The presence of cardiac remodeling at 24 and 25 years raises concern about previously unrecognized or longstanding exposure to

elevated blood pressure. However, neither patient had a documented previous diagnosis, and the duration of hypertension cannot be established from the available records.

Renal Involvement in Case 1

Case 1 demonstrated markedly elevated urea and creatinine, indicating severe renal dysfunction, with serum uric acid also markedly elevated at 786 $\mu\text{mol/L}$. A single abnormal renal-function measurement during an acute severe illness does not establish chronic kidney disease. Without previous results demonstrating persistent renal impairment, the more appropriate description is severe renal dysfunction or acute kidney injury, depending on the clinical course and baseline data available.

The renal abnormalities may have contributed to the difficulty in controlling blood pressure and may themselves have represented acute target-organ injury. Conversely, renal disease can contribute to secondary hypertension, illustrating the bidirectional relationship between kidney disease and hypertension [2,4].

Lifestyle Risk Factors

Both patients had modifiable risk factors. Case 1 was a heavy smoker, unemployed, reported poor sleep, and reported no regular exercise. Case 2 was a chronic smoker, reported heavy alcohol consumption, was unemployed, reported poor sleep, and reported no regular exercise.

Tobacco exposure, excessive alcohol consumption, physical inactivity, and unhealthy dietary patterns are established modifiable cardiovascular risk factors [1,4,7]. Lifestyle intervention, including smoking cessation, avoidance of excessive alcohol, increased physical activity, and dietary improvement, should form part of comprehensive hypertension management [1,2].

Unemployment, poor sleep, and lack of exercise are reported characteristics in these two cases; however, this two-case series cannot establish that these factors caused severe hypertension or intracerebral hemorrhage.

Family History

Case 1 had a maternal history of poorly controlled hypertension, whereas Case 2 had no reported family history. Family history is an important risk factor for hypertension, but absence of a family history does not exclude hypertension or secondary causes [2,7]. Young adults with severe hypertension should therefore undergo systematic assessment rather than having the condition attributed solely to familial or lifestyle factors.

Secondary Hypertension

The severity and very young age at presentation raise the possibility of secondary hypertension. The 2024 ESC Guidelines recommend considering secondary causes in young adults, including renal disease, primary aldosteronism, renovascular disease, endocrine disorders, medication- or substance-related hypertension, and obstructive sleep apnoea [2].

In Case 2, renal function, thyroid function, and abdominal ultrasonography were normal. These investigations do not exclude all secondary causes. For example, evaluation for primary aldosteronism generally requires appropriate aldosterone and renin testing, while suspected renovascular hypertension may require dedicated vascular imaging when clinically indicated [2].

In Case 1, the severity of the acute illness and poor clinical condition limited the opportunity for a comprehensive secondary-hypertension evaluation. Therefore, the aetiology of hypertension in both cases remains incompletely established, and the cases should not be classified definitively as primary hypertension.

Differences in Clinical Outcome

The outcomes differed substantially. Case 1 had impaired consciousness, severe renal dysfunction, pulmonary findings, suspected infection/sepsis, and progressive clinical deterioration, ultimately resulting in death after 18 days. Case 2 survived, achieved substantial blood pressure reduction, and showed improvement in left-sided muscle strength by one-month follow-up.

The different outcomes may reflect differences in the severity and extent of acute target-organ injury, systemic complications, and neurological status at presentation. Because this is a two-case series, no causal inference can be made regarding the factors responsible for the different outcomes.

The second case also demonstrates that normalization of blood pressure does not necessarily result in immediate neurological recovery. Cerebral injury from intracerebral hemorrhage may produce persistent deficits requiring prolonged rehabilitation, and neurological recovery can continue beyond the acute hospitalization.

Implications for Resource-Limited Settings

These cases are relevant to low- and middle-income settings, where the burden of hypertension is substantial and detection and control remain challenging [1]. WHO estimates that two-thirds of adults aged 30–79 years with hypertension live in low- and middle-income countries [1]. The 2025 WHO hypertension profile for Timor-Leste estimated a 2024 hypertension prevalence of 35% among adults aged 30–79 years and reported that only about 12% of adults with hypertension had controlled blood pressure [5].

Early detection of elevated blood pressure at primary healthcare level may provide an opportunity to initiate treatment before severe target-organ damage develops. Strengthening routine blood pressure screening, access to essential antihypertensive medicines, cardiovascular risk assessment, referral pathways, and long-term follow-up may therefore be particularly important in Timor-Leste [1,5].

Case 1 also illustrates the challenges associated with advanced renal and neurological disease, including consideration of renal replacement therapy and family involvement in treatment decisions. These observations are descriptive and should not be generalized beyond the individual cases.

Clinical Implications

- Blood pressure should be routinely measured in young adults, including those without a previous diagnosis of hypertension [1,2].
- Very severe hypertension in a young adult should prompt consideration of secondary hypertension rather than automatic attribution to primary hypertension [2].
- Assessment for hypertension-mediated organ damage should include renal function and urinalysis, ECG, and echocardiography where available [2].
- Acute neurological symptoms accompanied by severe hypertension require urgent neuroimaging to distinguish intracerebral hemorrhage from other causes of stroke [3].
- Smoking and excessive alcohol consumption should be actively addressed as part of cardiovascular risk reduction [1,4].
- Blood pressure control alone may not reverse established neurological injury; early and continued rehabilitation is important after stroke [3].
- Long-term follow-up is essential after intracerebral hemorrhage because persistent hypertension increases the risk of recurrent cerebrovascular events [3,4].

LIMITATIONS

This case series has several limitations. First, it describes only two patients and therefore cannot establish causal relationships or generalize outcomes to all young adults with hypertension. Second, the duration of hypertension before presentation was unknown in both patients. Third, evaluation for secondary

hypertension was incomplete, particularly in Case 1 because of the patient's critical condition. Fourth, detailed information regarding the timing and intensity of acute blood pressure management was incomplete. Finally, follow-up information for Case 2 was limited to one month after hospital discharge.

CONCLUSION

Severe hypertension can cause devastating complications even in very young adults. In this two-case series, two men aged 24 and 25 years presented with severe hypertension complicated by intracerebral hemorrhage and significant neurological impairment. Both demonstrated evidence of hypertensive cardiac target-organ damage, while one patient also had severe renal dysfunction and died during hospitalization.

The cases emphasize that hypertension in young adults should not be overlooked or considered benign. Early identification and confirmation of hypertension, assessment for secondary causes, evaluation of target-organ damage, appropriate blood pressure treatment, smoking cessation, reduction of harmful alcohol consumption, rehabilitation, and long-term follow-up are essential.

In resource-limited settings such as Timor-Leste, strengthening blood pressure screening, treatment, referral, and continuity of care at primary healthcare and community levels may help prevent premature cardiovascular, renal, and cerebrovascular complications.

Declarations

Ethics approval and consent to participate

Ethical approval for this case series was granted by the Executive Director of Hospital Nacional Guido Valadares (HNGV), Dili, Timor-Leste, and the approval was formally dispatched to the Department of Research, Education and Training for institutional research purposes. The study was conducted in accordance with institutional requirements governing retrospective clinical research. Patient-level information was treated as confidential institutional clinical data, and no personally identifiable information was disclosed in this manuscript.

Consent for publication

Informed consent for publication was discussed with the patients, and patient-identifying information was excluded from the manuscript.

Data availability

The clinical information used in this case series was obtained from institutional medical records. Patient-identifying information has been excluded from the manuscript. The underlying records are subject to institutional confidentiality requirements.

Competing interests

The authors declare no competing interests.

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Authors' contributions

Mateus Pinheiro and Julianti da Costa contributed to the study design, data collection, data analysis, manuscript preparation, and editing. The other authors contributed to data collection and clinical investigation according to their respective areas of expertise. All authors reviewed and approved the final manuscript.

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REFERENCES

1. World Health Organization. Hypertension. Geneva: World Health Organization; 2025. Available from: <https://www.who.int/news-room/fact-sheets/detail/hypertension>.
2. McEvoy JW, McCarthy CP, Bruno RM, Brouwers S, Canavan MD, Ceconi C, et al. 2024 ESC Guidelines for the management of elevated blood pressure and hypertension. *Eur Heart J*. 2024;45(38):3912–4018. doi:10.1093/eurheartj/ehae178.
3. Greenberg SM, Ziai WC, Cordonnier C, Dowlatshahi D, Francis B, Goldstein JN, et al. 2022 Guideline for the management of patients with spontaneous intracerebral hemorrhage: a guideline from the American Heart Association/American Stroke Association. *Stroke*. 2022;53(7):e282–e361. doi:10.1161/STR.0000000000000407.
4. Unger T, Borghi C, Charchar F, Khan NA, Poulter NR, Prabhakaran D, et al. 2020 International Society of Hypertension global hypertension practice guidelines. *Hypertension*. 2020;75(6):1334–1357.
5. World Health Organization. Global report on hypertension 2025: Timor-Leste hypertension profile. Geneva: World Health Organization; 2025.
6. Roth GA, Mensah GA, Johnson CO, Addolorato G, Ammirati E, Baddour LM, et al. Global burden of cardiovascular diseases and risk factors, 1990–2019: update from the GBD 2019 study. *J Am Coll Cardiol*. 2020;76(25):2982–3021.
7. Arnett DK, Blumenthal RS, Albert MA, Buroker AB, Goldberger ZD, Hahn EJ, et al. 2019 ACC/AHA guideline on the primary prevention of cardiovascular disease. *Circulation*. 2019;140(11): e596–e646.
8. Ettehad D, Emdin CA, Kiran A, Anderson SG, Callender T, Emberson J, et al. Blood pressure lowering for prevention of cardiovascular disease and death: a systematic review and meta-analysis. *Lancet*. 2016;387(10022):957–967.