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Endovascular recanalization of vertebral artery stump syndrome beyond 24 hours in a patient with medically refractory minor stroke: a case report

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Case Report

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Abstract

Background

Vertebral artery stump syndrome (VASS) is a rare cause of posterior circulation ischemic stroke and carries a high risk of recurrence, with a reported recurrent stroke rate of up to 37% with medical therapy alone. For patients with symptom onset beyond 24 hours, fluctuating progression, and refractoriness to medical treatment, the safety and efficacy of endovascular therapy beyond the conventional time window remain insufficiently supported by evidence. We report a case of medically refractory VASS treated successfully with endovascular recanalization 20 days after symptom onset.

Case presentation:

A 47-year-old man presented with sudden-onset dysarthria and right-sided limb weakness accompanied by dizziness, nausea, vomiting, and transient bilateral visual obscuration, and was admitted with dysarthria and right-sided hemisensory disturbance and weakness (National Institutes of Health Stroke Scale [NIHSS] score 4). Despite intravenous thrombolysis with tenecteplase and subsequent dual antiplatelet therapy, his symptoms fluctuated and progressed over 20 days, with recurrent infarctions involving the bilateral occipital lobes, left thalamus, right thalamus, and right temporo-occipital lobe. Digital subtraction angiography (DSA) revealed occlusion of the right vertebral artery origin with retrograde collateral flow distal to the occlusion, confirming VASS. Endovascular recanalization with aspiration thrombectomy, distal embolic protection, and balloon-expandable stenting of the right vertebral artery origin was performed 20 days after symptom onset, resulting in successful recanalization. At discharge (postoperative day 6), the NIHSS score was 1 and the modified Rankin Scale (mRS) score was 0. No stroke recurrence occurred during 12 months of follow-up.

Conclusions

Endovascular intervention may be a feasible and effective option for medically refractory mild VASS beyond the conventional time window, and this rare syndrome merits attention regarding its diagnostic criteria and the timing of endovascular treatment.

Background

Vertebral artery stump syndrome (VASS) is a rare cause of posterior circulation ischemic stroke, with a low incidence yet a high risk of recurrence [1, 2]. Medical therapy alone often fails to eliminate the source of embolism in patients with VASS, and endovascular intervention has emerged as an important treatment option [3]. Currently, there is insufficient evidence to support the safety and efficacy of endovascular therapy for VASS patients with symptom onset beyond 24 hours—or even longer—who exhibit fluctuating progression and refractoriness to medical therapy. We report a case of a patient with

mild VASS in whom the interval from symptom onset to procedure was as long as 20 days, with neurological symptom fluctuation and worsening during medical treatment, who achieved a favorable outcome after endovascular intervention.

Case presentation

A 47-year-old man was admitted to our hospital in July 2025 with a chief complaint of dysarthria and right-sided hemisensory disturbance and weakness lasting for one week. The patient experienced sudden-onset dysarthria and right-sided limb weakness, accompanied by dizziness, nausea, vomiting, and transient bilateral visual obscuration, one week prior to admission (July 2, 2025). He initially presented to a local hospital. Since symptom onset, his neurological symptoms had shown repeated exacerbation.

Past medical history: The patient had a 3-year history of hypertension without regular treatment. He had a clear family history of hypertension. There was no history of diabetes mellitus, coronary artery disease, cerebrovascular disease, or other medical conditions. He denied any history of smoking or alcohol consumption.

Physical examination on admission: Neurological examination revealed blunted responsiveness, non-fluent speech, and right-sided limb muscle strength of grade 3/5. The National Institutes of Health Stroke Scale (NIHSS) score was 4. No significant abnormalities were found on other systemic examinations.

Laboratory and imaging findings: Routine blood tests, liver and renal function, lipid profile, coagulation function, and autoimmune antibody panel were unremarkable. Electrocardiography and echocardiography (including Holter monitoring) showed no significant abnormalities. Pharmacogenetic testing indicated intermediate metabolism of clopidogrel (possible CYP2C19 loss-of-function alleles). Brain magnetic resonance imaging (MRI) performed on July 2, 2025, revealed acute infarction in the right occipital lobe (Fig. 1). Cervical vascular ultrasound showed hypoechoic material within the right vertebral artery, suggestive of thrombus. Cervical computed tomography angiography (CTA) performed in July 2025 demonstrated atherosclerosis with multiple mild stenoses; the right vertebral artery showed discontinuous opacification with multiple segments of severe stenosis or occlusion; and a fenestration anomaly of the intracranial segment of the left vertebral artery was noted (Fig. 2).

Diagnosis: (1) vertebral artery occlusion-related cerebral infarction; (2) hypertension, grade 3 (very high risk).

Clinical course: Based on the findings from the local hospital, the patient received intravenous thrombolysis with tenecteplase, following which his symptoms transiently improved. The next day (July 3, 2025), due to worsening dysarthria, repeat brain MRI revealed new infarcts in the bilateral occipital lobes and the left thalamus (Fig. 3). After admission to our hospital, given the intermediate metabolism of clopidogrel, the antiplatelet regimen was switched to ticagrelor (90 mg twice daily) combined with aspirin (100 mg daily), along with atorvastatin (20 mg daily) for intensive lipid-lowering therapy. On July

18, 2025, the patient experienced another episode of worsening dysarthria, accompanied by transient bilateral complete visual loss and left-sided motor disturbance. Repeat brain MRI showed new infarcts in the right thalamus and the right temporo-occipital lobe, with non-visualization of the right posterior cerebral artery (Fig. 4). Given the recurrent posterior circulation infarctions associated with vertebral artery occlusion, the clinical presentation was consistent with vertebral artery stump syndrome (VASS). Digital subtraction angiography (DSA) revealed occlusion of the right vertebral artery origin, severe stenosis at the origin of the right subclavian artery, and moderate stenosis at the origin of the left vertebral artery, with retrograde collateral flow distal to the occlusion, confirming the diagnosis of VASS (Fig. 5). Endovascular treatment was performed on July 22, 2025, 20 days after symptom onset. Aspiration thrombectomy using the ADAPT technique was performed under distal embolic protection, followed by balloon-expandable stent (Express SD, 5 mm × 19 mm) implantation at the origin of the right vertebral artery; post-procedural angiography demonstrated restored antegrade flow with good stent apposition (Fig. 5). Postoperatively, the patient continued dual antiplatelet therapy with aspirin (100 mg daily) and ticagrelor (90 mg twice daily) for 6 months, along with atorvastatin (20 mg daily). At discharge (postoperative day 6), the NIHSS score had decreased to 1, and the modified Rankin Scale (mRS) score was 0.

Follow-up: At 3, 6, and 12 months postoperatively, transcranial Doppler (TCD) and cervical vascular ultrasound demonstrated patent flow through the right vertebral artery stent, and no clinical stroke events occurred during the follow-up period. The timeline of clinical events is summarized in Table 1.

Table 1. Timeline of clinical events, imaging findings, and management

Date	Event
July 2, 2025	Sudden onset of dysarthria and right-sided limb weakness with transient bilateral visual obscuration; admission MRI showed acute right occipital lobe infarction (Fig. 1); CTA performed (Fig. 2)
July 3, 2025	Neurological deterioration; repeat MRI showed new infarcts in the bilateral occipital lobes and the left thalamus (Fig. 3)
July 18, 2025	Recurrent deterioration with transient bilateral complete visual loss and left-sided motor disturbance; repeat MRI showed new infarcts in the right thalamus and the right temporo-occipital lobe (Fig. 4)
July 22, 2025	DSA confirmed VASS (Fig. 5); endovascular recanalization with ADAPT aspiration thrombectomy, distal embolic protection, and balloon-expandable stent implantation at the origin of the right vertebral artery
Postoperative day 6	Discharge; NIHSS score 1; mRS score 0
3, 6, and 12 months postoperatively	TCD and cervical vascular ultrasound showed patent stent flow; no stroke recurrence

Discussion and Conclusions

Vertebral artery stump syndrome (VASS) results from the formation of a stump after occlusion of the vertebral artery origin, leading to blood stasis, secondary thrombus formation and embolization, causing distal embolism [4-6]. It accounts for approximately 1.4% of posterior circulation ischemic strokes [1]. Approximately one-third of patients experience stroke recurrence during hospitalization, and recurrence in the acute phase can be fatal [2]. In 2012, Kawano et al. reviewed the data of 3,463 patients with ischemic stroke and proposed the diagnostic criteria for VASS: (1) acute posterior circulation ischemic stroke; (2) occlusion of the origin of the vertebral artery confirmed by MRA, CTA, or DSA; (3) the presence of retrograde blood flow distal to the ipsilateral occluded vertebral artery; and (4) exclusion of other etiologies of stroke [2]. Subsequently, Zhang et al. further refined these criteria, requiring that the occlusion be located in the extracranial segment while the intracranial stump remains preserved, and that residual intracranial arterial stenosis be <30% after endovascular treatment [7]. The mechanisms by which VASS leads to acute ischemic stroke primarily include: (1) low-flow or turbulent collateral flow in the culprit vertebral artery, predisposing to thrombus formation; (2) direct embolism from turbulent flow distal to the occlusion; and (3) oscillatory blood flow resulting from collateral compensation and retrograde flow, leading to red thrombus formation and causing recurrent, multiple infarcts [8].

Regarding treatment, the management of VASS is divided into two main categories: medical therapy and endovascular intervention. Medical therapy primarily involves antiplatelet or anticoagulant agents. Meng et al. reported a case of VASS with recurrent posterior circulation infarction despite dual antiplatelet therapy; after switching to anticoagulation, no recurrence was observed during 9 months of follow-up [8]. Su et al. noted that antiplatelet agents have limited efficacy in preventing recurrent embolism in VASS [5]. Kawano et al. suggested that the low-flow or turbulent state in the vertebral artery predisposes to thrombus formation, and thus anticoagulation combined with dose adjustment may be the preferred strategy for preventing stroke recurrence; however, large-scale studies are lacking to confirm this [5]. Nevertheless, some patients with VASS continue to experience recurrent ischemic events despite optimal antithrombotic therapy [2,9,10]; medical therapy alone is often insufficient to eliminate the source of embolism, and the risk of stroke recurrence due to embolization from the stump persists. Therefore, endovascular intervention has become an important treatment option [2,3,9,10].

With the advancement of neurointerventional techniques, endovascular treatment has become an important therapeutic modality for VASS. In terms of timing, most studies advocate for early intervention after the first ischemic event, particularly in patients with a high risk of recurrence. Su et al. pointed out that when severe ischemia results from occlusion of the vertebral artery origin and medical therapy is ineffective, percutaneous transluminal angioplasty (PTA) and stent implantation may be considered [5]. The main therapeutic strategies include: (1) PTA/stenting to recanalize the occluded vertebral artery and restore antegrade flow, although there is a risk of distal embolization from stump thrombus during the procedure; (2) parent artery occlusion using coils or other embolic agents to seal the diseased vertebral artery and eliminate the stump, albeit at the cost of reducing part of the basilar artery blood supply; and (3) thrombectomy combined with stenting, in which aspiration thrombectomy is first performed to remove the stump and distal thrombus, followed by stent placement depending on the degree of residual stenosis [5]. Endovascular treatment of VASS has demonstrated certain efficacy [3,11-14]. Sakisuka et

al. reported three cases: one treated with balloon angioplasty followed by stent placement, and two treated with coil embolization of the distal segment of the occluded vertebral artery; no recurrence was observed during follow-up [1].

In the present case, DSA revealed occlusion of the right vertebral artery origin with retrograde flow distal to the occlusion, meeting the diagnostic criteria for VASS. Despite early intravenous thrombolysis and aggressive dual antiplatelet therapy combined with lipid-lowering therapy during the acute phase, the patient's symptoms continued to fluctuate and progress, suggesting that medical therapy alone was insufficient to control the disease. Endovascular intervention was performed 20 days after symptom onset, and the patient achieved a favorable clinical outcome, with no stroke recurrence during the 1-year follow-up period. The main limitation of this report is that it describes a single patient, and the findings cannot be generalized; multicenter studies with larger cohorts are needed to confirm the safety and efficacy of endovascular treatment in this setting.

In conclusion, for patients with VASS who are refractory to medical therapy and have a persistent risk of embolism, endovascular treatment administered beyond the 24-hour window may represent an effective therapeutic option.

Abbreviations

Abbreviation	Full form
ADAPT	A Direct Aspiration First-Pass Technique
CTA	computed tomography angiography
CYP2C19	cytochrome P450 2C19
DSA	digital subtraction angiography
DWI	diffusion-weighted imaging
MRA	magnetic resonance angiography
MRI	magnetic resonance imaging
mRS	modified Rankin Scale
NIHSS	National Institutes of Health Stroke Scale
PTA	percutaneous transluminal angioplasty
T2 FLAIR	T2 fluid-attenuated inversion recovery
TCD	transcranial Doppler
VASS	vertebral artery stump syndrome

Declarations

Ethics approval and consent to participate

This study was approved by the Institutional Review Board of The 960 Hospital of People's Liberation Army (approval number: (2026) Research Ethics Review No. (135)). Written informed consent was obtained from the patient.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Availability of data and materials

Data sharing is not applicable to this article as no datasets were generated or analysed during the current study.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

Concept and design: Xianxian Guo, Huaiqiang Hu. Analysis and interpretation: Xinxin Yang, Shuai Miao, Junbin Yin. Data collection: Xianxian Guo, Xinxin Yang. Writing the article: Xianxian Guo, Xinxin Yang. Critical revision of the article: Huaiqiang Hu, Xinxin Yang, Junbin Yin. Final approval of the article: Huaiqiang Hu, Xianxian Guo. All authors read and approved the final manuscript.

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Figures

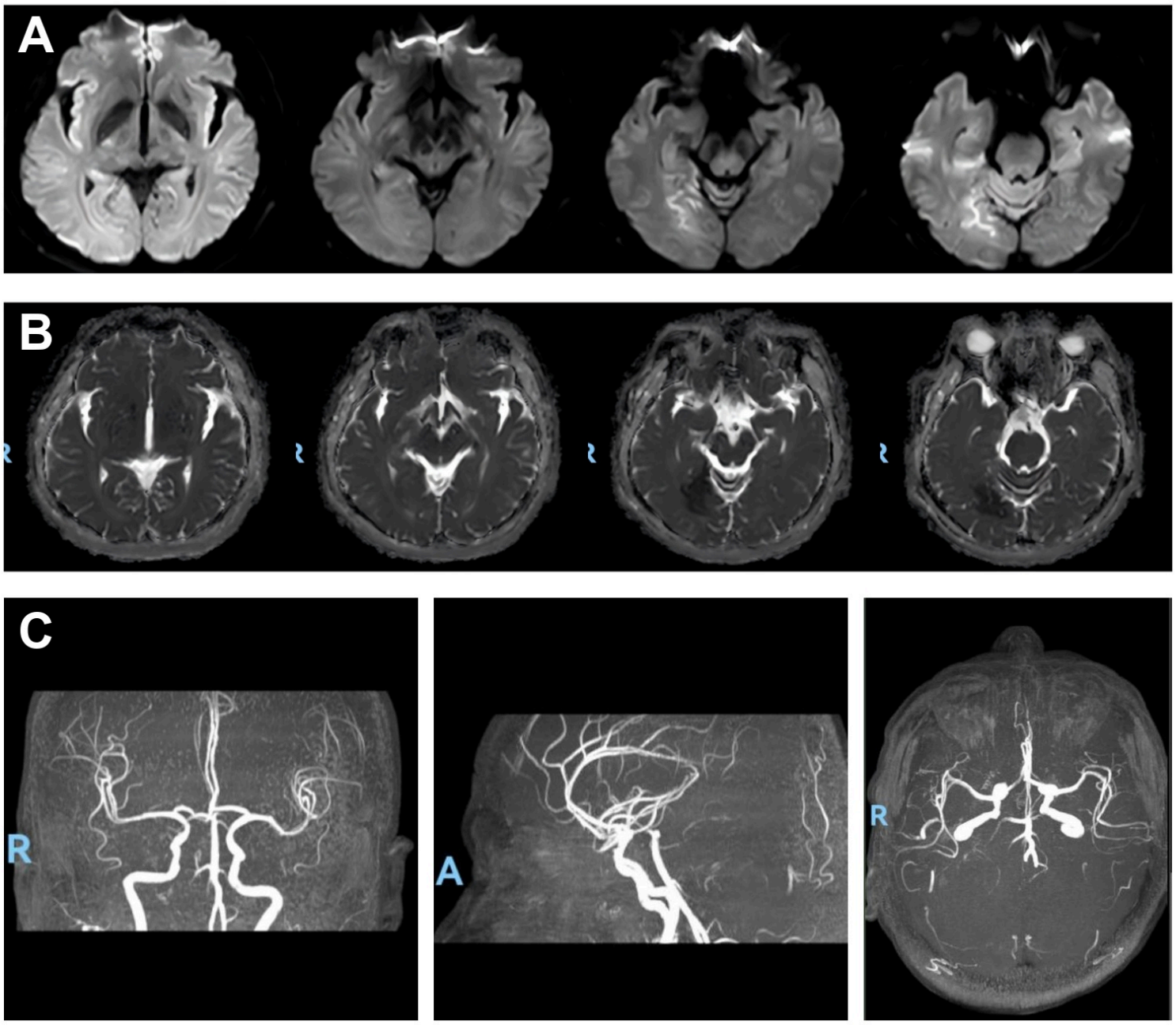


Figure 1

Emergency brain magnetic resonance imaging (MRI) performed on July 2, 2025. (A) Diffusion-weighted imaging (DWI) sequence; (B) T2-weighted sequence; (C) Magnetic resonance angiography (MRA). The

findings were collectively suggestive of acute cerebral infarction in the right occipital lobe, with poor visualization of the bilateral posterior cerebral arteries.

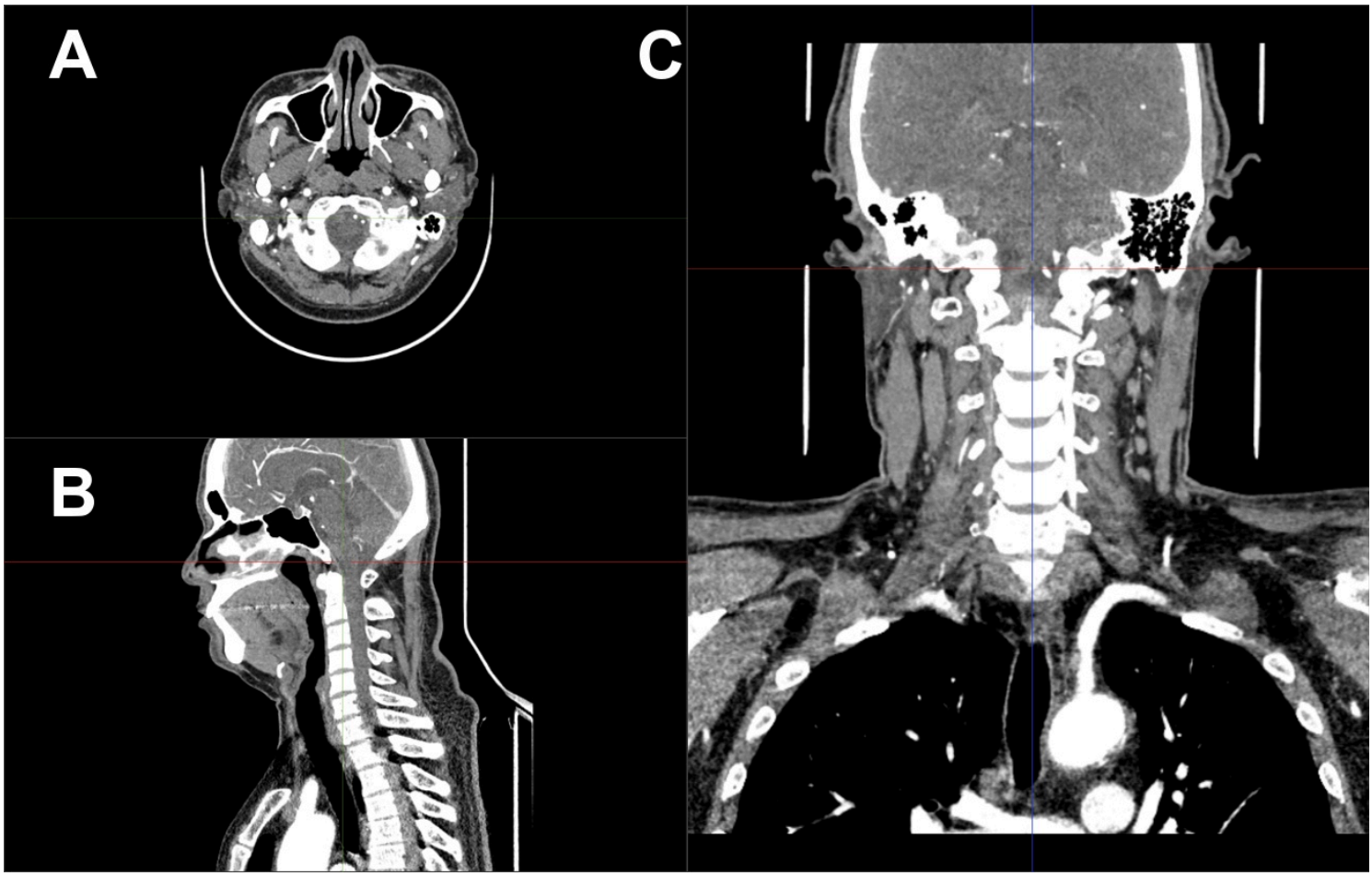


Figure 2

Three-dimensional reconstruction of head and neck computed tomography angiography (CTA). (A) Axial, (B) sagittal, and (C) coronal views show discontinuous opacification of the right vertebral artery, with multiple segments of severe stenosis or occlusion.

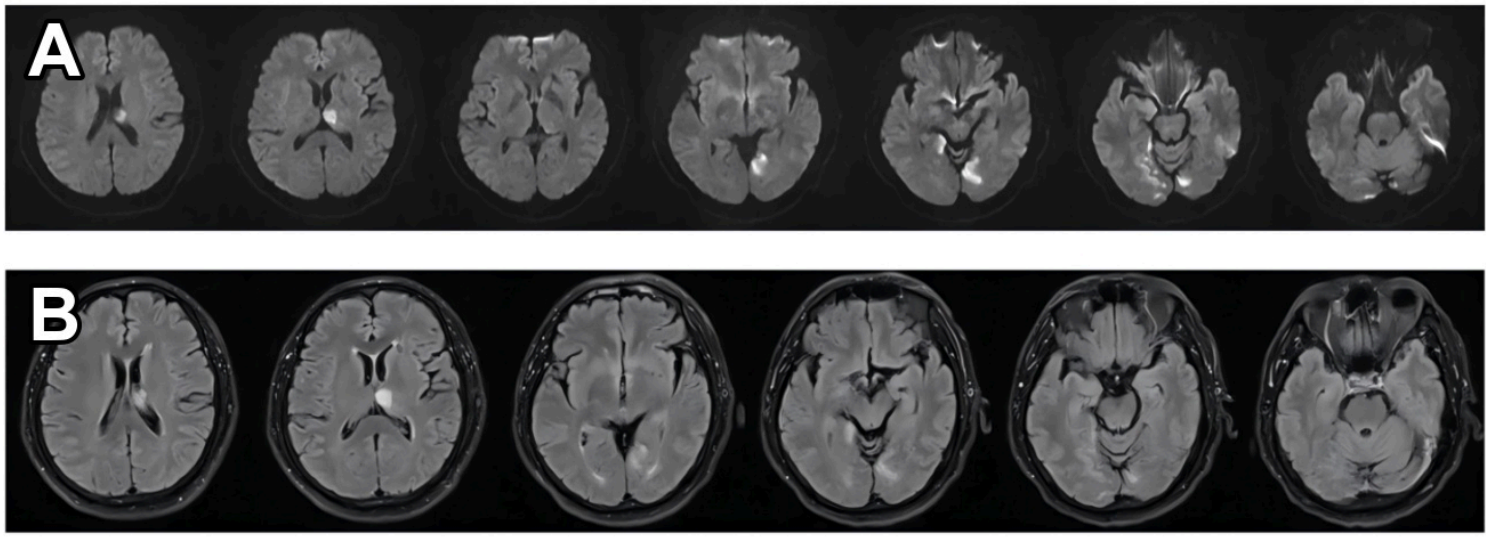


Figure 3

Brain magnetic resonance imaging (MRI) performed on July 3, 2025, following neurological deterioration. (A) Diffusion-weighted imaging (DWI) sequence; (B) T2 fluid-attenuated inversion recovery (T2 FLAIR) sequence. The findings were collectively suggestive of acute cerebral infarction in the bilateral occipital lobes and the left thalamus.

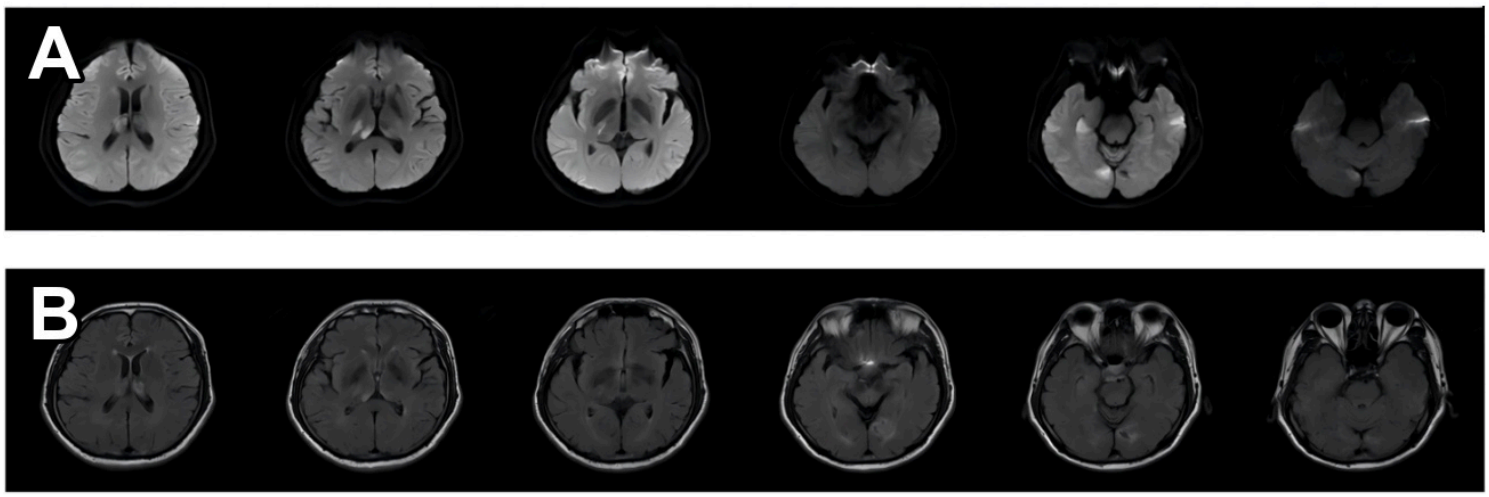


Figure 4

Brain magnetic resonance imaging (MRI) performed on July 18, 2025, following neurological deterioration. (A) Diffusion-weighted imaging (DWI) sequence; (B) T2 fluid-attenuated inversion recovery (T2 FLAIR) sequence. The findings were collectively suggestive of new infarcts in the right thalamus and the right temporo-occipital lobe, with non-visualization of the right posterior cerebral artery.

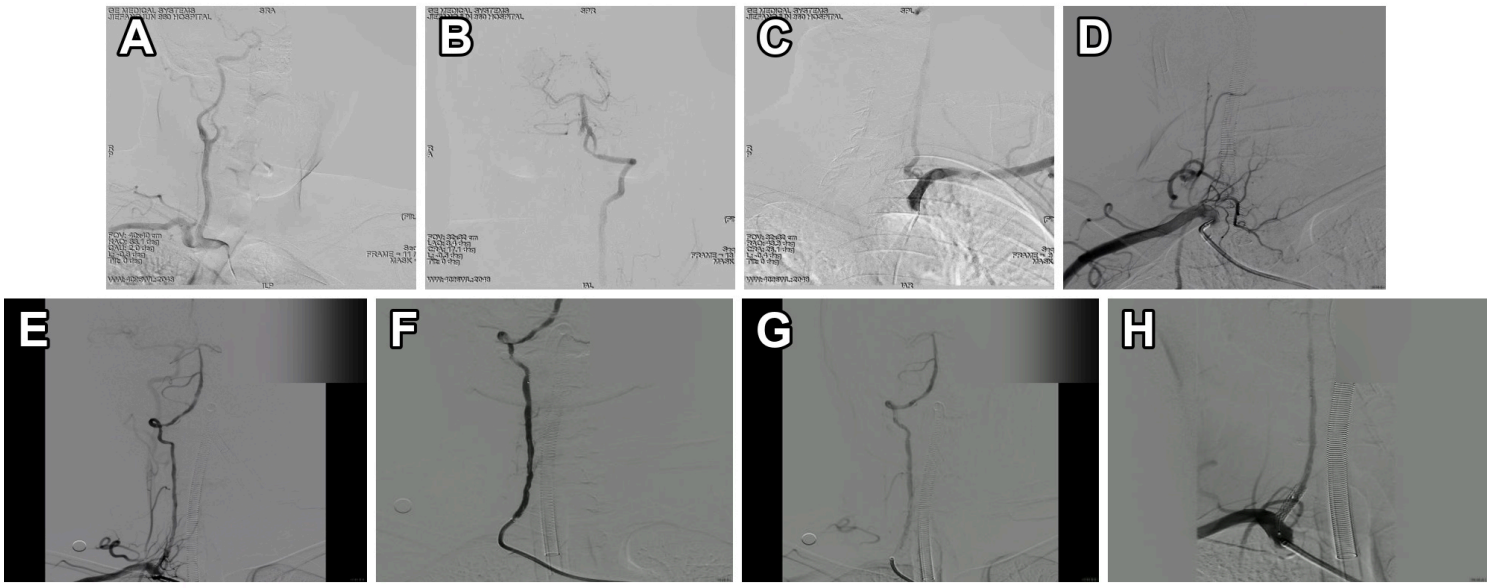


Figure 5

Digital subtraction angiography (DSA) and endovascular treatment. (A) Occlusion of the right vertebral artery; (B) severe stenosis at the origin of the right subclavian artery; (C) moderate stenosis at the origin of the left vertebral artery. Endovascular procedure: (D) collateral flow from the right ascending cervical artery; (E) angiography after thrombectomy using the ADAPT technique showed residual thrombus signals in the V1 and V2 segments; (F) angiography after re-deployment of a distal protection device demonstrated patent antegrade flow; (G) and (H) implantation of a balloon-expandable stent (Express SD, 5 mm × 19 mm) at the origin of the right vertebral artery; post-deployment angiography showed good stent apposition with patent antegrade flow.