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Case Report

Reversal of Progressive In-Hospital Posterior Artery-to-Artery Embolic Stroke With Argatroban: A Case Report

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Abstract

Aims/Background: In-hospital progressive stroke in patients with pre-existing large artery occlusion presents a therapeutic dilemma, particularly when standard reperfusion strategies are unsuitable. This case report aims to illustrate a mechanism-guided medical approach in this complex scenario. **Case Presentation**: We report the case of a 65-year-old male patient with chronic left vertebral artery occlusion, hospitalised for dizziness, who experienced acute neurological deterioration with a National Institutes of Health Stroke Scale (NIHSS) score ranging from 2 to 14. Magnetic resonance imaging confirmed a new pontine infarction. Endovascular therapy was deferred due to suspected artery-to-artery embolism from an unstable plaque. Instead, argatroban infusion combined with antiplatelet therapy was initiated within 5 h. **Results**: The patient showed gradual improvement, and follow-up imaging demonstrated complete thrombus resolution. The NIHSS score was reduced to 4 at discharge. **Conclusion**: This case highlights the successful use of a mechanism-guided approach with argatroban for thrombolysis-naïve posterior circulation stroke, effectively balancing the thrombotic and haemorrhagic risks. This suggests that personalised anticoagulation may optimise outcomes in similar complex scenarios in which standard reperfusion is unsuitable.

Keywords: ischemic stroke; vertebral artery; anticoagulants; case reports

1. Introduction

In-hospital embolic strokes, representing 6.5%–15.0% of all strokes [1], pose a considerable clinical challenge. This is due to difficulties in definitive diagnosis, partly because advanced techniques such as non-obstructive general angioscopy with high diagnostic accuracy [2] have not yet been widely adopted, in addition to a lack of consensus on optimal management strategies for specific subtypes. The management of progressive in-hospital ischaemic stroke in patients with preexisting large artery occlusive disease presents a critical therapeutic challenge. The underlying pathophysiology is often an artery-to-artery embolism from unstable plaques [3]. While endovascular therapy (EVT) is standard intervention for community-onset large vessel occlusion, its role in hospitalized patients with neurological deterioration due to non-occlusive thrombosis remains poorly defined, creating a significant evidence and guidance gap [4,5]. Argatroban, a direct thrombin inhibitor with rapid onset and minimal renal dependence, is a promising candidate [6]. When combined with antiplatelet therapy, it achieves a synergistic effect by concurrently targeting both coagulation and platelet pathways [7]. This multipathway inhibition enhances efficacy while maintaining a favourable balance with bleeding risk [8], a hallmark of the mechanism of action of argatroban. Herein, we report a unique case of progressive posterior circulation stroke caused by an artery-to-artery embolism that was successfully managed using this mechanism-guided combination therapy, thereby filling a critical gap in the literature and providing a practical framework for an antithrombotic strategy in this complex scenario.

2. Case Report

A 65-year-old male farmer was admitted on 12 August 2022 with a five-year history of recurrent dizziness triggered by activity or postural changes, accompanied by profuse sweating and resolving spontaneously within minutes. His medical history revealed pulmonary tuberculosis treated with streptomycin a decade ago, which resulted in hearing impairment. He had no history of hypertension, diabetes mellitus, dyslipidaemia, stroke, or transient ischaemic attack. He was a nonsmoker and denied regular alcohol intake. His home medications included betahistine mesylate (6 mg orally [PO] three times a day [TID]; H20040130, Eisai Pharmaceutical Co., Ltd., Suzhou, China) and mecobalamin (0.5 mg PO TID; H20080102, Beijing Si-Huan Pharmaceutical Co., Ltd., Beijing, China). There was no remarkable family history of cardiovascular or neurological disease.

One month prior to admission (18 July 2022), he experienced a sudden loss of consciousness during an activity, resulting in head trauma. An external hospital evaluation

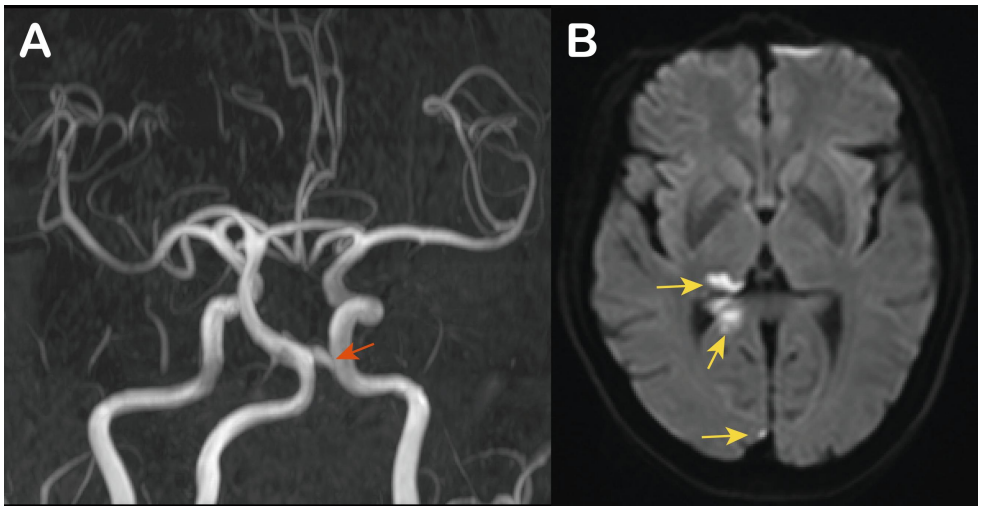

Fig. 1. Acute imaging. (A) Magnetic resonance angiography on admission showed left vertebral artery occlusion (red arrow). (B) The patient had acute cerebral infarctions (yellow arrows) in the right dorsal thalamus, the right side of the splenium of the corpus callosum, and the right occipital lobe on diffusion-weighted imaging sequences obtained on admission.

revealed a cervical accessory fracture and spinal cord injury. Cervical computed tomography angiography (CTA) showed occlusion of the left vertebral artery segments V1–V3.

On admission, his vital signs were stable: blood pressure 114/72 mmHg, heart rate 72 beats per minute, respiratory rate 18 breaths per minute, and body temperature 36.7 °C. His height, weight, and body mass index were 177 cm, 65 kg, and 20.8 kg/m^2, respectively. Upon admission, neurological examination revealed clear consciousness, a left pupil diameter of 3.5 mm (sluggish light reflex) versus 2.0 mm (brisk) on the right, a left visual field deficit, and decreased superficial sensation in the left upper limb; however, no homonymous hemianopia or signs of ataxia were observed during careful testing. The National Institutes of Health Stroke Scale (NIHSS) score was 2. Preliminary diagnoses included left vertebral artery occlusion, syncope under investigation, and cerebral infarction. Magnetic resonance angiography (MRA) performed on admission confirmed occlusion of the left vertebral artery (Fig. 1A). Diffusion-weighted imaging (DWI) revealed multiple recent cerebral infarctions in the right dorsal thalamus, right side of the splenium of the corpus callosum, and right occipital lobe (Fig. 1B). The initial treatment consisted of clopidogrel (75 mg PO once daily (QD); H20056410, Sanofi (Hangzhou) Pharmaceutical Co., Ltd., Hangzhou, China), rosuvastatin (10 mg PO QD; HJ20160611, IPR Pharmaceuticals, INCORPORATED, Canóvanas, Puerto Rico), betahistine (6 mg PO TID), and mecobalamin (0.5 mg PO TID). Routine gastric protection and fluid replacement were also administered.

On hospital day 3 (15 August), the patient developed acute loss of consciousness and left hemiplegia. Neurological examination revealed stupor, moderate dysarthria, and left limb muscle strength of grade 0–1, elevating the NIHSS score to 14. An emergency magnetic resonance imaging (MRI)-DWI sequence demonstrated a suspicious high signal intensity in the right cerebral peduncle, suggestive of an acute infarction (Fig. 2A). Following a thorough discussion, the team opted against EVT. This decision was based on two key considerations: First, the deterioration occurred within 24 hours of the initial infarction diagnosis, suggesting "infarct progression" rather than a new embolic event. Second, given the known chronic left vertebral artery occlusion, the aetiology was suspected to be artery-to-artery embolism from an unstable plaque. We hypothesised that such thrombi might be more "non-occlusive" or "friable" than the typical cardioembolic clots. Consequently, the treatment goals shifted to stabilising the culprit lesion and inhibiting thrombus propagation. Intensified anticoagulation with argatroban infusion (10 mg intravenously (IV) every 4 hours (Q4H), targeting an activated partial thromboplastin time of 60–80 s; H20227090, Hunan Sailong Pharmaceutical (Changsha) Co., Ltd., Changsha, China) was initiated within 5 h of symptom onset, supplemented with alprostadil (10 µg IV QD; H10980024, Beijing Tide Pharmaceutical Co., Ltd., Beijing, China) to improve microcirculation. Clopidogrel was concomitantly discontinued.

The patient's neurological deficits gradually improved after treatment. Repeat MRA on 17 August indicated thrombus formation at the basilar tip (Fig. 2B), and the corresponding DWI sequences confirmed new acute infarctions in the right pontine (Fig. 2C) and red nucleus (Fig. 2D). In response to the evidence of disease progression, the antiplatelet regimen was intensified by resuming clopidogrel (75 mg PO QD) in addition to ongoing aspirin (HJ20160684, Bayer HealthCare Manufacturing S.r.l, Canovanas, Puerto Rico) and argatroban. The argatroban dose was simultaneously adjusted to 10 mg intravenously every 12 h, strictly following the manufacturer's guidelines.

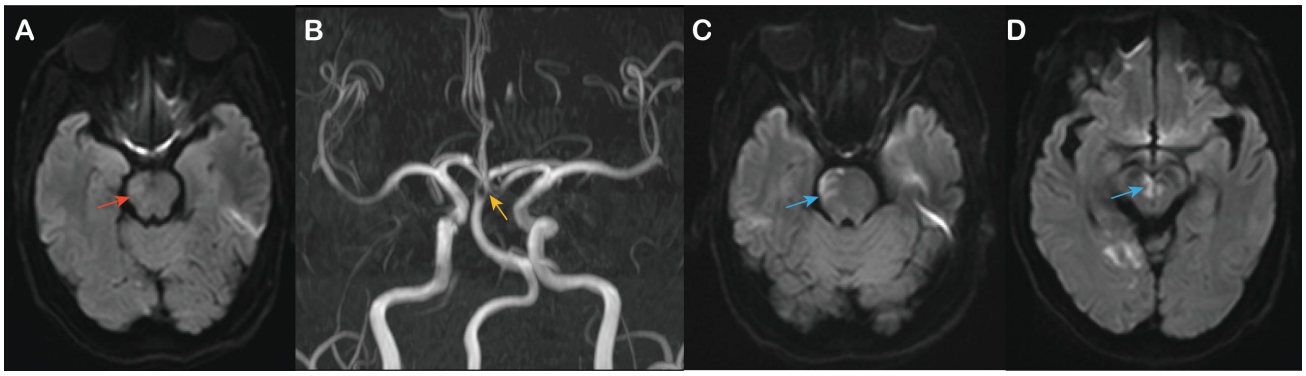

Fig. 2. Neuroimagings following in-hospital neurological deterioration. (A) Re-examination of diffusion-weighted imaging (DWI) sequences in 5 h after loss of consciousness during hospitalization suggested an acute infarction in the right cerebral peduncle (red arrow). (B) Magnetic resonance angiography on 17 August showed a newly-formed thromboembolic occlusion of the top of the basilar artery (yellow arrow). (C,D) DWI sequences on 17 August confirmed new acute infarctions in the right pontine and the red nucleus (blue arrows).

Follow-up head and neck CTA on 22 August showed complete resolution of the basilar tip thrombus and achieved vascular recanalization (Fig. 3). Dual antiplatelet therapy, alprostadil, and statins were continued for secondary prevention until the day of discharge. By the time of discharge on 24 August, the patient was conscious, able to eat orally, and had left limb muscle strength recovered to grade 4, with an NIHSS score of 4.

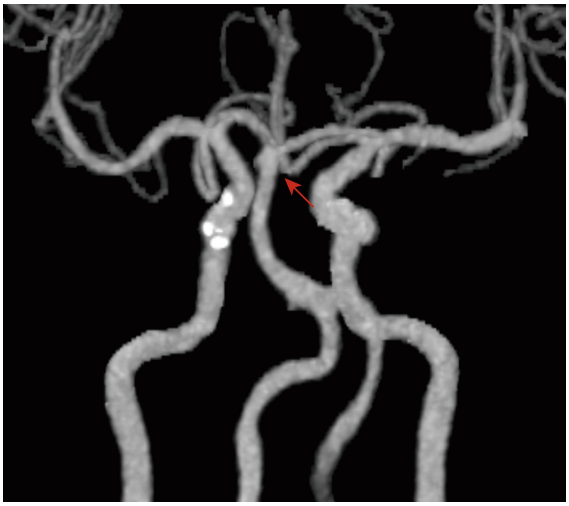

Fig. 3. Post-treatment vascular recanalization. After 7 days of argatroban therapy, head and neck computed tomography angiography shows disappearance of the thromboembolic occlusion of the top of the basilar artery (red arrow).

Long-term follow-up information was obtained via telephone on 26 November 2025. The patient reported no further cerebrovascular events since discharge and expressed satisfaction with the clinical outcomes. He remained functionally independent in daily living activities and was ambulatory without assistance, although he acknowledged a mild residual deficit compared with his pre-stroke baseline. He has been attending regular follow-up visits at a local hospital, as advised.

The CARE Checklist has been attached as **Supplementary Material** associated with this article.

3. Discussion

This case of in-hospital progressive posterior circulation stroke resulting from an artery-to-artery embolism exemplifies a clinical scenario in which standard reperfusion therapies are often contraindicated. In our thrombolysis-naïve patient with chronic vertebral occlusion, the administration of argatroban plus antiplatelet therapy led to significant clinical improvement, as corroborated by imaging-confirmed thrombus resolution. This outcome highlights the potential of mechanism-guided anticoagulation therapy in selected patients.

The efficacy of argatroban is highly context-dependent. Its adjunctive use following intravenous thrombolysis (with or without thrombectomy) has shown no benefit and increased mortality in trials such as Multi-Arm Optimization of Stroke Thrombolysis (MOST) [9] and a 2024 New England Journal of Medicine (NEJM) study [10]; however, a distinct therapeutic niche exists for thrombolysis-naïve patients exhibiting active thrombotic progression. This is supported by a randomised trial in which argatroban added to antiplatelet therapy significantly improved 90-day functional outcomes in patients with early neurological deterioration [7]. Similarly, dual-pathway inhibition (antiplatelet and anticoagulant) can be effective in branch atheromatous diseases when monotherapy fails [11]. Our case epitomises the latter scenario, characterised by high thrombotic activity and a low perceived risk of haemorrhagic transformation, which collectively established a favourable risk-benefit profile for argatroban.

Mechanistically, the observed embolic regression may be attributed to the dual action of argatroban in inhibiting thrombin-mediated clot propagation and enhancing endogenous fibrinolysis [12]. Prompt initiation of therapy within 5 h of deterioration is critical, likely enabling intervention within the crucial window for thrombus stabilisation. Consequently, this case reinforces the value of a precision medicine approach for stroke management. This illustrates that, for a subset of patients with progressive stroke due to artery-to-artery embolism in the absence of large-vessel occlusion, targeted anticoagulation may present a more viable therapeutic strategy than mechanical reperfusion. Future studies should seek to validate this pathophysiological stratification and optimise individualised antithrombotic strategies.

The principal strength of this case lies in its timely illustration of a successful mechanism-guided medical strategy for complex in-hospital stroke scenarios. However, this study had several limitations. Only official reports, rather than initial CTA images, from the patient's local hospital were available. This restricts a baseline visualisation of the patient's vasculopathy. Although the proposed mechanism of enhanced endogenous fibrinolysis is supported by the existing literature, we did not report temporal changes in specific blood indices (e.g., serial tissue plasminogen activator or D-dimer levels) to directly confirm fibrinolysis activation in this case. And outcomes were assessed primarily using the NIHSS, which may not have fully captured details of posterior circulation stroke recovery. In addition, as this is a single case report, the conclusions may be uncertain and require validation through controlled studies.

4. Conclusion

This case demonstrates that argatroban combined with antiplatelet therapy can effectively treat progressive in-hospital posterior circulation stroke caused by artery-to-artery embolism, particularly in thrombolysis-naïve patients with a high thrombotic but low haemorrhagic risk. This underscores the importance of a mechanism-guided individualised approach beyond standard reperfusion strategies.

Learning Points

• In-hospital progressive posterior circulation stroke secondary to artery-to-artery embolism is a distinct clinical scenario in which standard reperfusion therapies are often contraindicated and require pathophysiology-guided alternatives.

• For thrombolysis-naïve patients with active thrombotic progression, a combination of argatroban and antiplatelet therapy represents a highly effective pharmacological strategy for stabilising nonocclusive thrombi and facilitating clinical recovery.

• The efficacy of argatroban is context-dependent, underscoring the importance of a precision medicine approach that carefully balances the individual thrombotic drive against the haemorrhagic risk.

• This case elucidates a practical clinical framework for patient selection, highlighting the ideal candidates for targeted anticoagulation, such as those with a high thrombotic burden, low haemorrhagic risk, and early neurological deterioration in the absence of prior thrombolysis or thrombectomy.

• Collectively, these findings advocate a paradigm shift toward mechanism-based treatment personalisation beyond conventional reperfusion, pointing to the need for future research focused on biomarker-guided patient stratification for therapies such as argatroban.

Availability of Data and Materials

The data supporting the findings of this study are included in the article. Additional data are available from the corresponding author upon reasonable request.

Author Contributions

YTS made substantial contributions to the study conception and design, and was responsible for data validation, visualization, and drafting the original manuscript. FW contributed to the methodology development and conducted the formal investigation. QG and YXS participated in the data validation process. DYC made substantial contributions to the study conception and design, and was involved in manuscript review and editing, supervised the project, acquired funding, and provided administrative support. All authors contributed to the important editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

The study was conducted in strict accordance with the ethical principles outlined in the Declaration of Helsinki. This case report was approved by the Human Research Ethics Committee of the Zhejiang University School of Medicine (Approval Number: I2025266). Informed consent for publication was obtained from the patient.

Acknowledgment

Not applicable.

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Conflict of Interest

The authors declare no conflict of interest.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at https://doi.org/10.31083/BJHM52084.

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