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Transient Ischemic Attack Progressing to Top-of-the-Basilar Syndrome: A Case Report

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Patient: Male, 77-year-old
Final Diagnosis: Top-of-the-basilar syndrome
Symptoms: Right hemiplegia • transient coma
Clinical Procedure: —
Specialty: Neurology

Objective: Unusual clinical course

Background: Top-of-the-basilar syndrome (TOBS) is a rare but clinically heterogeneous posterior circulation disorder characterized by acute ischemia affecting 2 or more territories supplied by the distal basilar artery, including the thalami, midbrain, occipital lobes, medial temporal lobes, and cerebellum. Intravenous thrombolysis with recombinant tissue plasminogen activator (rt-PA) is an established treatment for eligible patients with TOBS, and reports of transient coma induced during rt-PA infusion remain exceedingly scarce.

Case Report: A 77-year-old man with non-valvular atrial fibrillation and hypertension presented with transient visual hallucinations—initially diagnosed as a transient ischemic attack. Emergency head and neck computed tomography angiography revealed severe stenosis-occlusion of the left posterior cerebral artery P1 segment. Within 12 hours, he developed dysarthria and right hemiparesis, prompting intravenous thrombolysis with rt-PA (0.9 mg/kg). Fifty minutes after infusion, he experienced abrupt, self-limiting loss of consciousness, without evidence of intracranial hemorrhage on emergent computed tomography. Subsequent magnetic resonance imaging confirmed incomplete TOBS, showing acute infarcts in bilateral thalami, left hippocampus, and right cerebellar hemisphere. Magnetic resoance angiography demonstrated resolution of the prior P1 stenosis but new suspicious narrowing in the distal P2 segment.

Conclusions: This case suggests that the clinical symptoms of cardiogenic TOBS change rapidly and are diverse. This diversity can occur before and during intravenous thrombolytic therapy. If neurological deterioration occurs during the use of thrombolytic agents, it is necessary to promptly identify whether it is stroke progression or hemorrhagic transformation.

Keywords: stroke • thrombolytic therapy • symptom assessment

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/953007

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Introduction

Classical top-of-the-basilar syndrome (TOBS) was first characterized by Louis R. Caplan in 1980, who defined it as a distinct posterior circulation disorder resulting from ischemia at the distal bifurcation of the basilar artery [1]. Its clinical presentation is highly heterogeneous and classically includes visual field deficits, vertical or conjugate gaze palsy, altered consciousness, memory impairment, and visual hallucinations, reflecting involvement of the thalami, midbrain, occipital cortices, and medial temporal lobes [1,2]. Contemporary diagnostic criteria emphasize multimodal neuroimaging confirmation, particularly diffusion-weighted magnetic resonance imaging (MRI) demonstrating 2 or more acute ischemic lesions within territories supplied by the distal basilar artery branches (eg, posterior cerebral, superior cerebellar, and posterior inferior cerebellar arteries) [2]. Intravenous thrombolysis with recombinant tissue plasminogen activator (rt-PA) has demonstrated efficacy in selected TOBS cases [3,4], and documented instances of acute, transient coma occurring during rt-PA infusion remain rare; however, we highlight an underappreciated potential complication of reperfusion therapy in this syndrome. We report a case of a patient who progressed from transient ischemic attack (TIA) to incomplete TOBS. During intravenous thrombolysis, the patient experienced transient coma. This clinical phenomenon has been rarely reported in the past and is worthy of attention from doctors making decisions on intravenous thrombolysis use.

Case Report

A 77-year-old man with a documented history of non-valvular atrial fibrillation and hypertension presented to the emergency department at night with acute-onset visual hallucinations—lasting approximately 30 minutes and resolving spontaneously—without associated dysarthria, limb weakness, or altered consciousness. Neurological examination was unremarkable. Non-contrast head computed tomography (CT, **Figure 1A-1D**) revealed a patchy low-density shadow in the right frontal lobe (considering an old infarct). Head and neck computed tomography angiography (CTA, **Figure 1E-1H**) demonstrated severe stenosis-occlusion of the left posterior cerebral artery P1 segment. Routine laboratory investigations, including complete blood count, renal function with electrolytes, and coagulation profile, were within the reference range. Based on clinical and imaging findings, the patient was diagnosed with TIA and admitted for secondary prevention. Dual antiplatelet therapy (aspirin 100 mg daily and clopidogrel 75 mg daily) and high-intensity statin therapy (atorvastatin 20 mg nightly) were initiated immediately.

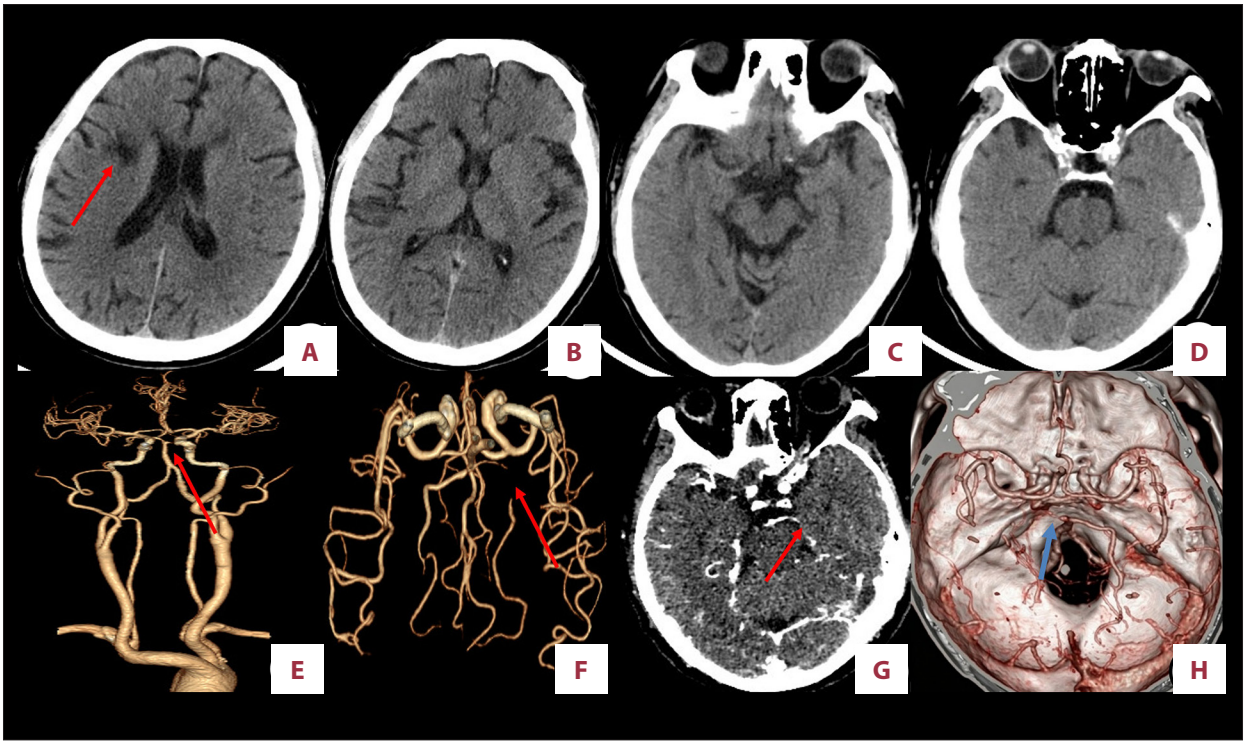

Figure 1. (**A**) The red arrow indicates a patchy low-density shadow in the right frontal lobe (considering an old infarct). (**B-D**) No obvious abnormal lesions were found in the bilateral thalami, midbrain, and pons. (**E-H**) Computed tomography angiography reveals severe stenosis to occlusion in the P1 segment of the left posterior cerebral artery.

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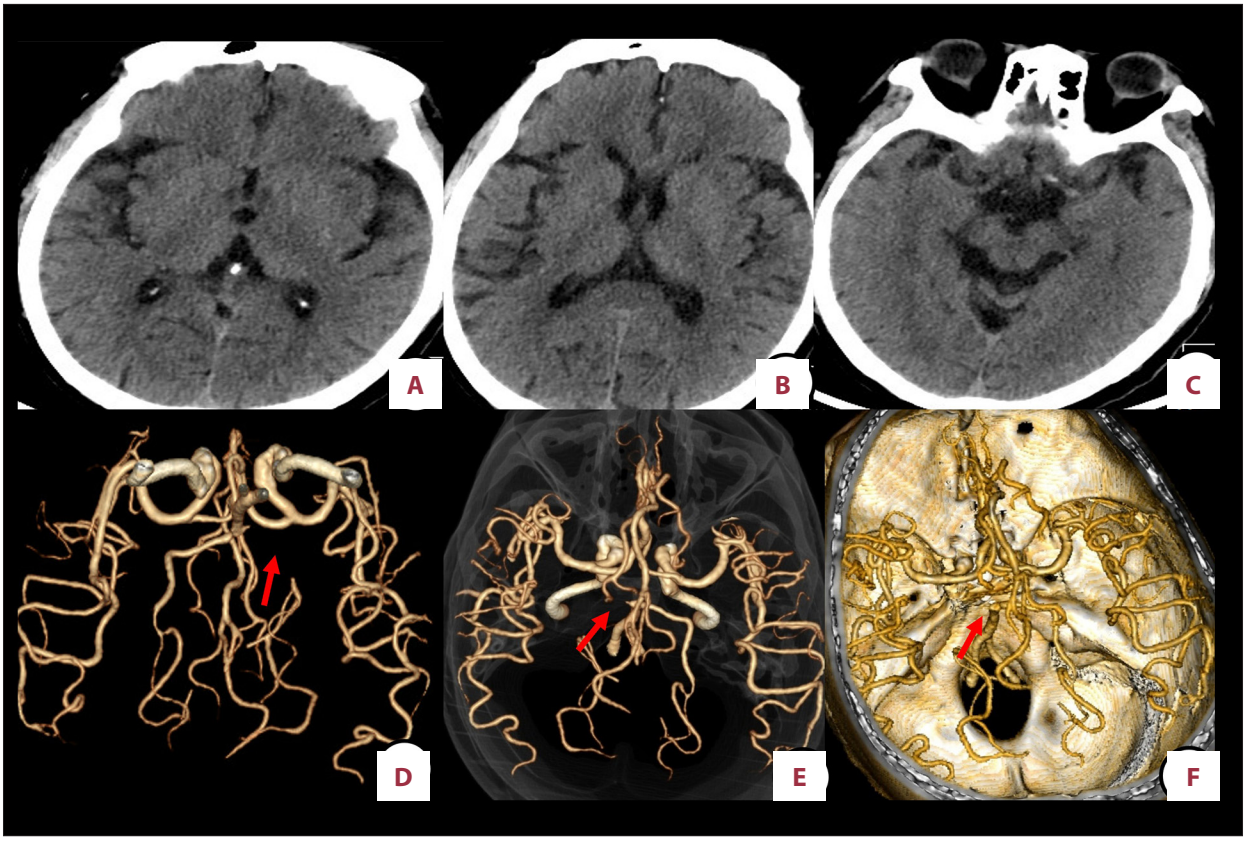

Figure 2. (**A-C**) No obvious abnormal lesions were found in the bilateral thalami and midbrain. (**D-F**) Computed tomography angiography reveals severe stenosis to occlusion in the P1 segment of the left posterior cerebral artery.

Approximately 12 hours after admission, the patient developed acute right-sided hemiparesis and dysarthria. On examination, he exhibited right central facial palsy, right lingual palsy, right upper and lower limb muscle strength graded 3/5, and contralateral (right-sided) facial and limb hypoalgesia. Since the patient had non-valvular atrial fibrillation, in order to check for large vessel embolism and indications for endovascular interventional therapy, head CT and head and neck CTA examinations were performed again. Repeat non–contrast-enhanced head CT (**Figure 2A-2C**) showed no new hemorrhage or infarction. Follow-up CTA (**Figure 2D-2F**) confirmed persistent left posterior cerebral artery P1 stenosis-occlusion. Given the clear progression from TIA to acute ischemic stroke within the therapeutic window, intravenous thrombolysis with alteplase (0.9 mg/kg) was administered. Fifty minutes into the infusion, the patient experienced abrupt, transient loss of consciousness, which lasted less than 5 minutes, with spontaneous return to full alertness during transport to emergent cranial CT. That scan excluded intracranial hemorrhage. Notably, right limb strength improved to 4/5 coincident with awakening.

On hospital day 2, MRI of the brain demonstrated acute infarcts in the bilateral thalami, left hippocampus, and right cerebellar hemisphere (**Figure 3A-3D**). Magnetic resonance angiography (MRA, **Figure 3E, 3F**) revealed resolution of the prior left posterior cerebral artery P1 stenosis but new suspicious narrowing in the distal P2 segment. Antiplatelet agents were discontinued, and anticoagulation with dabigatran (110 mg twice daily) was initiated. By hospital day 10, right limb strength improved to 4+/5, and the modified Rankin Scale score was 2. At 3-month outpatient follow-up, neurological recovery was sustained, with the modified Rankin Scale score improving to 1.

Discussion

Classical TOBS is defined as acute ischemia affecting 2 or more territories supplied by the distal basilar artery branches, including the thalami, midbrain, occipital cortices, medial temporal lobes, and superior cerebellum, typically manifesting on diffusion-weighted MRI as 2 or more acute infarcts in these regions [2]. Clinical manifestations reflect this anatomical distribution and can include vertical gaze palsy, visual field deficits, altered consciousness, memory disturbance, ataxia, and hemisensory-motor deficits [2].

In the present case, the diagnosis of incomplete TOBS was confirmed by multimodal MRI, which demonstrated acute

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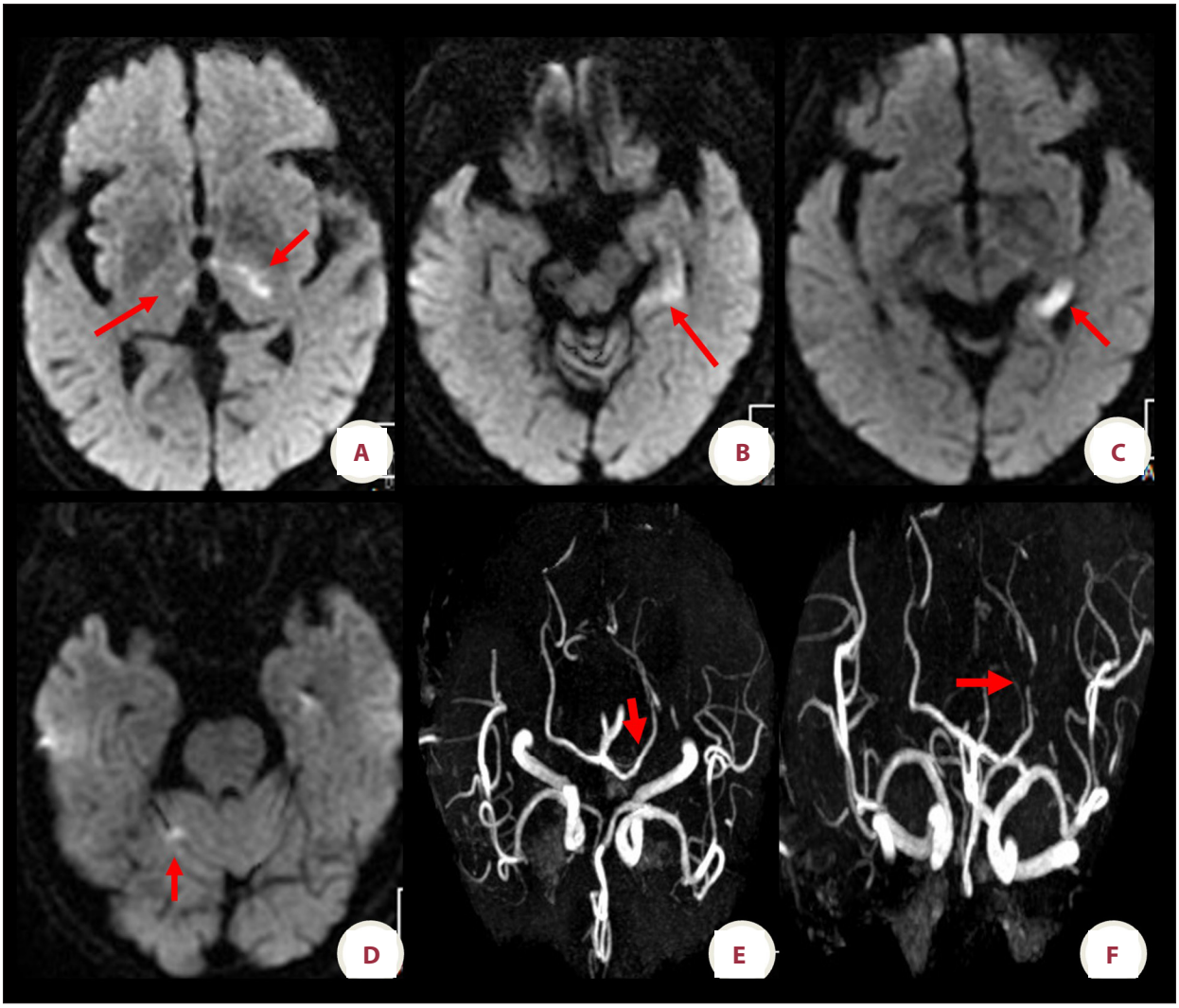

Figure 3. (**A**) Diffusion-weighted imaging reveals acute infarct lesions involving the bilateral thalamus. (**B, C**) The arrowhead indicates the site of acute infarction in the left hippocampus. (**D**) The arrow points to a punctate acute infarction focus in the right cerebellum. (**E**) Magnetic resonance angiography (MRA) reveals no stenosis in the P1 segment of the left posterior cerebral artery. (**F**) MRA reveals a suspicious stenosis at the distal end of the P2 segment of the left posterior cerebral artery.

infarcts involving the bilateral thalami, left hippocampus, and right cerebellar hemisphere. Unlike classical TOBS, the patient did not exhibit infarction of the midbrain or occipital lobes. Therefore, this presentation is more appropriately classified as incomplete TOBS.

This case highlights 2 pathophysiologically important questions: (1) what drives the rapid progression from TIA to multifocal cerebral infarction, and (2) why the patient experienced a transient coma. At admission, head-and-neck CTA revealed severe stenosis-occlusion of the left posterior cerebral artery P1 segment. Following intravenous thrombolysis, MRA showed complete resolution of the P1 lesion but newly apparent narrowing in the distal P2 segment. Given the patient's non-valvular atrial fibrillation, the initial P1 occlusion was highly likely cardiogenic. We propose that rt-PA–mediated thrombus fragmentation generated microemboli that migrated distally, causing secondary occlusion in the P2 territory—a mechanism consistent with documented patterns of embolic showering in posterior circulation syndromes [5,6]. If the infarct area of the bilateral thalami is in the area supplied by the bilateral paramedian arteries, it can lead to confusion, somnolence, stupor, and even coma [7]. According to the anatomical characteristics of the thalamus, the infarct site of the bilateral thalami in this patient was in the area supplied by the bilateral paramedian arteries. We speculated that the ischemic volume of this area was relatively large when the patient was in a coma.

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A transient coma was observed in this patient 50 minutes after the infusion of rt-PA, which is considered to be related to acute ischemia in both thalami. There are 2 possible mechanisms for bilateral thalamic infarction in this patient. First, the Percheron artery arose from the P1 segment of the left posterior cerebral artery in this patient, which is responsible for the blood supply to both dorsal thalami [8,9]. After thrombolytic therapy, the disintegration of emboli led to infarction in the bilateral thalamic regions, mainly on the left side. However, most Percheron artery infarctions usually involve both midbrains simultaneously [8], but this patient was not affected. Second, cardiogenic small emboli caused infarction in the right thalamus and right cerebellum, and the disintegration of emboli at the stenosis of the P1 segment of the left posterior cerebral artery caused infarction in the left thalamus and hippocampal region [10]. Regardless of the above mechanisms, the distribution of infarction involving multiple regions, such as both thalami, the left hippocampal region, and the right cerebellum, in this patient is consistent with the diagnosis of incomplete TOBS [2].

Conclusions

This case suggests that cardiogenic TOBS has diverse and rapidly changing clinical symptoms. If neurological deterioration (such as sudden disturbance of consciousness) occurs during the treatment with thrombolytic agents, clinicians need to make a rapid decision on whether it is symptomatic hemorrhagic transformation or stroke progression.

Department and Institution Where Work Was Done

Jiangyin People's Hospital Affiliated to Nantong University, Jiangyin, Jiangsu, PR China.

Patient Permission

Written informed consent was obtained from the patient for participation in the study.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

References:

1. Caplan LR. "Top of the basilar" syndrome. Neurology.1980;30(1):72-79
2. Ahn SH, Kim BJ, Kim YJ, et al. Patterns and outcomes of the top of the basilar artery syndrome: The role of the posterior communicating artery. Cerebrovasc Dis. 2018;46(3-4):108-17
3. Yan S, Zhou Y, Zhao Y, et al. Effect of imaging markers on reperfusion therapy in basilar artery occlusion. Ann Neurol. 2022;92(1):97-106
4. Lieschke F, Rauch M, Roller B, et al. Symptoms, imaging features, treatment decisions, and outcomes of patients with top of the basilar artery syndrome: Experiences from a Comprehensive Stroke Center. Neurocrit Care. 2025;43(1):69-79
5. Mattle HP, Arnold M, Lindsberg PJ, et al. Basilar artery occlusion. Lancet Neurol. 2011;10(11):1002-14
6. Salerno A, Strambo D, Nannoni S, et al. Patterns of ischemic posterior circulation strokes: A clinical, anatomical, and radiological review. Int J Stroke. 2022;17(7):714-22
7. Schmahmann JD. Vascular syndromes of the thalamus. Stroke. 2003;34(9):2264-78
8. Saltz G, Bruno A, Nichols F. Artery of Percheron stroke. Ann Neurol. 2024;95(4):800-1
9. Bordes S, Werner C, Mathkour M, et al. Arterial supply of the thalamus: A comprehensive review. World Neurosurg. 2020;137:310-18
10. Johnson AC. Hippocampal vascular supply and its role in vascular cognitive impairment. Stroke. 2023;54(3):673-85

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