

Endovascular intervention for bilateral paramedian thalamic stroke due to occlusion of the unilateral P1 segment of the posterior cerebral artery: illustrative cases

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BACKGROUND Occlusion of the unilateral P1 segment can result in bilateral paramedian thalamic infarction in patients with anatomical variants of the bilateral paramedian thalamic artery arising from a single P1 segment. Despite the life-threatening presentation of bilateral paramedian thalamic stroke, timely diagnosis is often challenging.

OBSERVATIONS The authors herein describe 3 patients treated with endovascular intervention for occlusion of the unilateral P1 segment wherein the bilateral paramedian thalamic arteries arose. All patients were admitted to the authors' emergency department with sudden-onset coma and respiratory distress; however, initial computed tomography was unremarkable. Despite suspicion of basilar artery occlusion, vertebral and carotid angiography revealed occlusion of the unilateral P1 segment. All patients were successfully treated with endovascular intervention. Overall, 2 patients had favorable outcomes (modified Rankin scale [mRS] scores of 0 and 1), whereas in 1 patient, the mRS score reached a baseline score of 3.

LESSONS In patients with the variant of the bilateral paramedian thalamic artery arising from a single P1 segment, occlusion of the unilateral P1 segment can be life threatening; nevertheless, timely endovascular treatment is effective. Carotid and vertebral angiography, rather than magnetic resonance or computed tomography angiography, is useful for immediate and reliable diagnosis of the relatively small vascular lesions.

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KEYWORDS endovascular intervention; paramedian thalamic stroke; P1 segment; posterior cerebral artery

The paramedian thalamic arteries are perforator branches arising from the P1 segment of the posterior cerebral artery (PCA) and supplying the paramedian thalamus and rostral midbrain. Although the overall prevalence rate of anatomical variants in which the paramedian thalamic arteries supplying the bilateral paramedian thalamus arise from a single P1 segment remains unknown,^{1,2} in the patients with this variant, occlusion of the unilateral P1 segment can cause bilateral paramedian thalamic infarction with or without midbrain involvement.^{1,3-5} In contrast to classic stroke manifestation, paramedian thalamic infarction has been notable for exhibiting consciousness impairment (from drowsiness to deep coma), respiratory dysfunction, and ophthalmological and neuropsychological signs, with

or without motor weakness, all of which are notable as prominent basilar artery syndromes.⁶ However, if basilar artery occlusion is not observed on vascular imaging studies, bilateral paramedian thalamic infarction caused by the occlusion of the unilateral P1 segment could remain undiagnosed; this is especially true in acute stroke intervention settings requiring timely diagnosis despite the severe outcome.⁶⁻⁸ Awareness of both the variants of paramedian thalamic arteries and the clinical presentation of occlusion of such variants is needed for prompt diagnosis and intervention.

Herein, we describe 3 patients who were successfully treated with endovascular intervention for occlusion of the unilateral P1 segment giving rise to the bilateral paramedian thalamic arteries.

ABBREVIATIONS AOP = artery of Percheron; CT = computed tomography; DTP = door-to-puncture; GCS = Glasgow Coma Scale; MR = magnetic resonance; MRI = magnetic resonance imaging; mRS = modified Rankin scale; NIHSS = National Institutes of Health Stroke Scale; OTD = onset-to-door; PCA = posterior cerebral artery; PCoA = posterior communicating artery; PTR = puncture-to-recanalization.

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We also discuss the utility of carotid as well as vertebral angiography for reliable diagnosis and the optimal treatment in the acute stroke setting.

Illustrative Cases

Case 1

A 63-year-old woman presented with the sudden onset of impaired consciousness and respiratory distress. On arrival, her Glasgow Coma Scale (GCS) and National Institutes of Health Stroke Scale (NIHSS) scores were 6 and 30, respectively. There was no definite ischemic change or hemorrhage on initial brain computed tomography (CT); in addition, basic laboratory test results were unremarkable. These findings raised suspicion of basilar artery syndrome, and emergency digital subtraction angiography was directly performed without any other imaging. The onset-to-door (OTD) and door-to-puncture (DTP) times were 240 minutes and 60 minutes, respectively. Because of the unspecific stroke symptom, intravenous thrombolysis was not performed. The left

vertebral angiogram showed the absence of the left P1 segment and occlusion of the right P2 segment (Fig. 1A). The left P2 segment was slightly filled via the left posterior communicating artery (PCoA) on the left common carotid artery (Fig. 1B). On the basis of clinical suspicion of bilateral paramedian thalamic ischemia due to occlusion of the left P1 segment, a mechanical thrombectomy was performed using the EMBOTRAP II stent retriever (Johnson & Johnson) and Catalyst 6 aspiration catheter (Stryker) (Fig. 1C). A Thrombolysis in Cerebral Infarction score of 2b was achieved after the first pass, with a puncture-to-recanalization (PTR) time of 32 minutes (Fig. 1D). The patient's consciousness and respiratory dysfunction improved immediately after recanalization. Postoperative brain magnetic resonance (MR) imaging (MRI) revealed a high-intensity area in the bilateral paramedian thalamic and occipital lobes, suggesting occlusion of the bilateral paramedian thalamic arteries (Fig. 1E). She was transferred to another hospital for rehabilitation due to vertical gaze palsy and attention deficit, and she was eventually discharged from that hospital with a modified Rankin scale (mRS) score of 1 at 3 months after stroke onset.

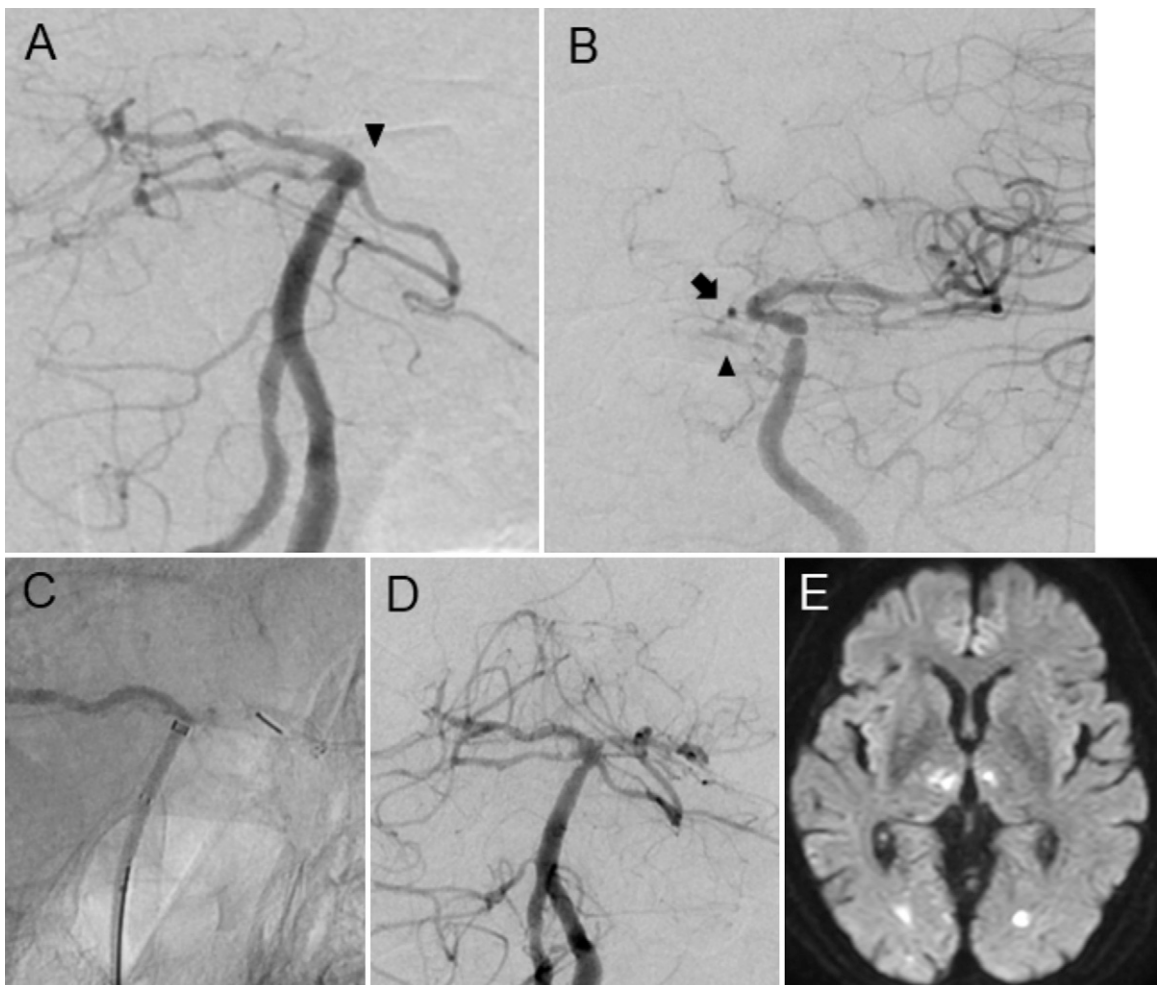


FIG. 1. Case 1. **A:** Left vertebral contrast injection showing a filling defect of the left PCA (*arrowhead*). **B:** Left common carotid contrast injection showing faint filling of the PCA (*arrowhead*) via the PCoA (*arrow*). **C:** A stent retriever deployed covering the left P1 segment through the aspiration catheter. **D:** Left vertebral contrast injection immediately after the thrombectomy showing recanalization of the left PCA. **E:** Diffusion-weighted MRI performed on postoperative day 4 reveals high-intensity areas in the bilateral paramedian thalamic and occipital lobes.

Case 2

A 73-year-old man presented with the sudden onset of deep coma with tetraparesis. On arrival, his GCS and NIHSS scores were 6 and 32, respectively. However, his impaired consciousness and motor weakness fluctuated in the emergency department. Brain CT findings and laboratory test results were unremarkable. The patient directly underwent emergent angiography with OTD and DTP times of 90 minutes and 120 minutes, respectively. Intravenous thrombolysis was performed with a door-to-needle time of 112 minutes. The left vertebral contrast injection showed stagnant flow in the left P1 segment (Fig. 2A). The left PCA was not depicted via the PCoA on the left common carotid contrast injection, suggesting occlusion at the P1–2 segment (Fig. 2B). On the basis of the patient's clinical symptoms, ischemia of the bilateral paramedian thalamic artery was suspected, and intravenous thrombolysis and endovascular treatment were performed. The left vertebral angiogram obtained immediately after crossing the occluded segment with a microguidewire showed complete recanalization of the left PCA and visualization of the bilateral supply of the paramedian thalamic artery from the left P1 segment (PTR time 12 minutes) (Fig. 2C). After recanalization, the coma and tetraplegia resolved rapidly. Postoperative brain MRI revealed no recent infarctions (Fig. 2D), and the postoperative course was uneventful. The patient was discharged with an mRS score of 0.

Case 3

A 75-year-old woman with a history of cardioembolic cerebellar infarction presented with a sudden onset of coma, respiratory dysfunction, and bilateral mydriasis. On arrival, her GCS and NIHSS scores were 3 and 32, respectively. The patient was promptly intubated in the emergency department. Brain CT findings and laboratory test results were unremarkable. On the basis of suspicion of basilar artery occlusion, emergency angiography was directly performed (OTD time 15 hours, DTP time 90 minutes). Because the patient arrived beyond the time window, intravenous thrombolysis was not performed. On vertebral angiography, a small stump was suspected at the basilar artery distal to the origin of the bilateral superior cerebellar arteries (Fig. 3A). The right carotid contrast

injection demonstrated the PCA distal to the P2 segment through the PCoA, which was smaller in size than its downstream PCA (Fig. 3B). The left carotid contrast injection demonstrated the PCA distal to the P2 segment through a funnel-shaped and sizable PCoA (Fig. 3C). The angiographic results of both the right and left carotid contrast injections suggested that the right P1 segment was not originally hypoplastic. On the basis of these findings, mechanical thrombectomy for isolated occlusion of the right P1 segment was performed by a combined technique using the Trevo NXT stent retriever (3 × 32 mm; Stryker) and the AXS Catalyst 6 aspiration catheter (Fig. 3D). After a second pass, complete recanalization of the right P1 segment and artery of Percheron (AOP) was achieved with a PTR of 130 minutes (Fig. 3E). Contrast staining of the bilateral medial thalami, midbrain, and pons was identified on postoperative noncontrast brain CT, indicating an ischemic insult in the bilateral paramedian thalami (Fig. 3F). However, no high-intensity area was observed on diffusion-weighted imaging performed 3 days after the intervention (Fig. 3G). Postoperatively, the patient regained consciousness. Mydriasis completely resolved within 24 hours after endovascular treatment. Although transient tracheostomy was required because of laryngeal edema, the patient's condition improved to a baseline mRS score of 3 at 1 month after symptom onset.

Discussion

Although PCA occlusion was excluded from previous randomized clinical trials on mechanical thrombectomy in acute ischemic stroke, mechanical thrombectomy is more often performed in the PCA occlusion by retrospective studies, demonstrating favorable angiographic and clinical outcomes.^{9,10} However, the severity of the symptoms can differ, depending on the occluded segment of the PCA. In fact, a recent study of patients with PCA occlusion demonstrated that the ischemic lesion involving the thalamus-midbrain was a predictor for unfavorable outcome after mechanical thrombectomy.¹¹ Paramedian thalamic arteries supplying the thalamus and hypothalamus, with or without supplying the midbrain, arise from the P1 segment of the PCA. Percheron described four variants of the paramedian thalamic arteries as follows: type I, the

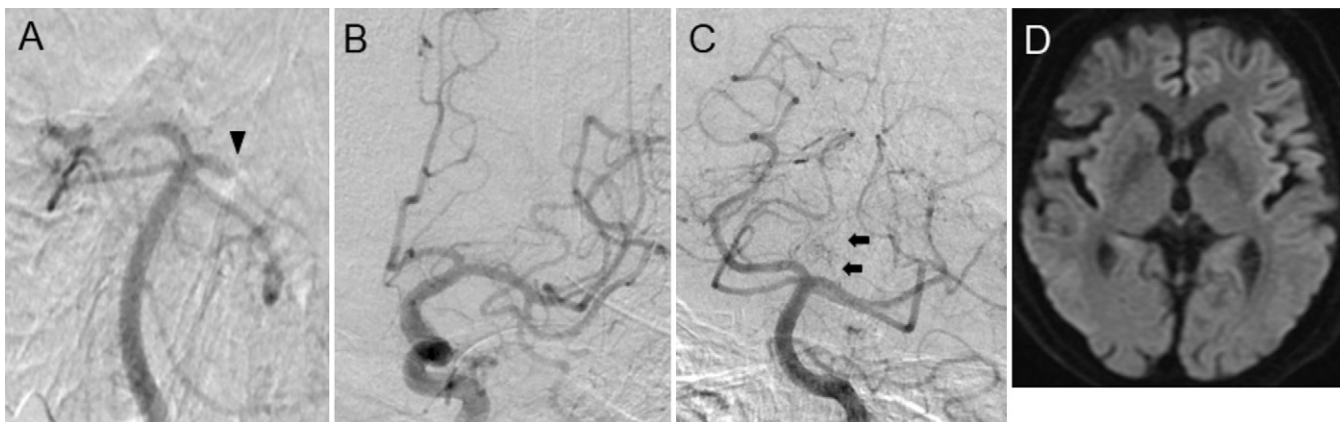


FIG. 2. Case 2. **A:** Left vertebral contrast injection showing a filling defect of the first segment of the left PCA (arrowhead). **B:** Left carotid injection demonstrating no filling of the left PCA via the PCoA. **C:** Left vertebral angiogram acquired immediately after the endovascular maneuver, showing recanalization of the left PCA and solitary paramedian thalamic artery arising from the left P1 segment (arrows). **D:** Diffusion-weighted MRI performed on postoperative day 3 revealing no ischemic lesion in the bilateral medial thalami.

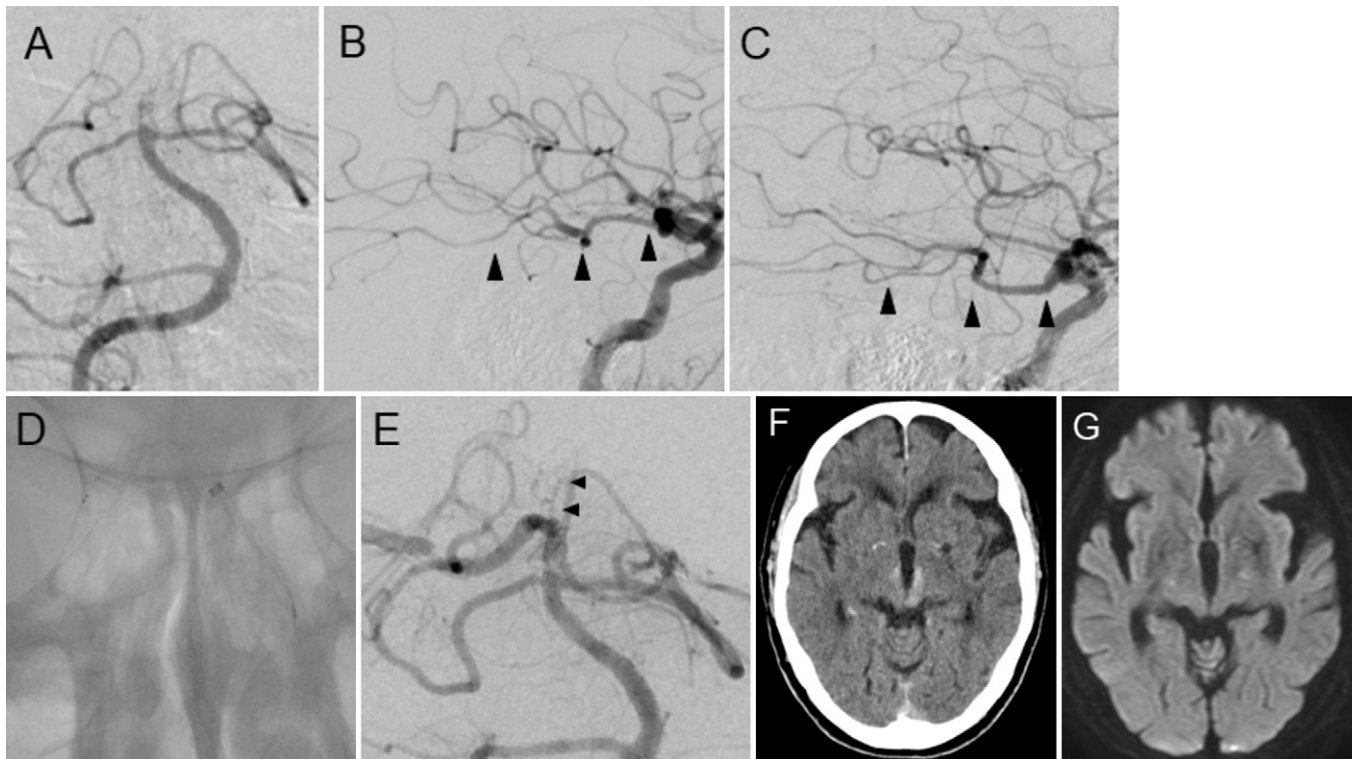


FIG. 3. Case 3. **A:** Right vertebral contrast injection showing small stump of the basilar artery distal to the bilateral superior cerebellar arteries. **B:** Right common carotid contrast injection showing the PCA via the relatively small PCoA (*arrowheads*). **C:** Left common carotid contrast injection showing the fetal-type PCA (*arrowheads*), suggesting the presence of the right P1 segment. **D:** A stent retriever was deployed, covering the right P1 segment through the aspiration catheter. **E:** Left vertebral injection immediately after thrombectomy showing recanalization of the right PCA and solid visualization of the bilateral paramedian thalamic arteries arising from the right P1 segment (*arrowheads*). **F:** Postoperative noncontrast CT showing contrast staining in bilateral medial thalami. **G:** Diffusion-weighted MRI performed on postoperative day 3 revealing no ischemic lesion in bilateral medial thalami.

most common—each perforating artery arises from bilateral PCA; type IIa, the perforating arteries arise directly from a single P1 segment; type IIb, the truncal perforating artery, also called the AOP, arises from a single P1 segment; and type III, several small perforating branches arise from the arc bridging the right and left P1 segments.^{2,4,5} In patients with type IIa or IIb variants, occlusion of the unilateral P1 can cause bilateral paramedian thalamic stroke. Patients with bilateral paramedian thalamic ischemia often greatly manifest basilar artery syndrome, such as coma and respiratory distress, with or without hemiparesis. However, if the basilar artery appears patent on CT angiography or magnetic resonance angiography, the diagnosis might be delayed and misinterpreted by physicians, especially nonneurologists; hence, only conservative management might be administered.^{6,12}

Observations

In this study, all 3 patients manifested deep coma. Overall, 2 patients (cases 1 and 3) had respiratory distress and required respiratory support before endovascular intervention, and 1 patient (case 3) had bilateral mydriasis. Therefore, although the basilar artery is opacified in patients presenting with the top of the basilar artery syndrome, further investigation of bilateral paramedian thalamic ischemia should be performed.

To evaluate the occlusion of the P1 segment of the bilateral paramedian thalamic artery, we performed angiography after screening for impaired consciousness, including basic laboratory testing and a noncontrast whole-body CT scan. Before the endovascular treatment,

all patients underwent vertebral contrast injection, followed by carotid contrast injection, ipsilateral to the affected P1 segment; this is because the potential competitive flow between P1 and PCoA might make it difficult to ascertain whether the nonopacified P1 segment on the vertebral injection indicates apparently aplastic/hypoplasticity or occlusion of the P1 segment. In cases 1 and 2, based on the findings of both vertebral and carotid injections, occlusion of the P1 segment was confirmed. In case 3, although it remained unclear whether the P1 segment was apparently aplastic/hypoplastic or occluded on the basis of vertebral injection, the smaller caliber of the right PCoA relative to that of the P2 segment and the large funnel-shaped left PCoA implied that the right P1 was originally patent and then acutely occluded. Therefore, in acute ischemic stroke settings, when the top of the basilar artery syndrome is suspected on the basis of clinical symptoms, but if the basilar artery appears patent on vertebral injection, both the vertebral and carotid injections are useful for investigating true occlusion of the P1 segment from apparent aplastic/hypoplasticity.

MR and/or CT angiography may help detect PCA occlusion involving the P1 segment. However, because motion artifacts on MR and/or CT angiography caused by respiratory distress in cases 1 and 3 or fluctuating consciousness disturbance in case 2 were of concern, only selective angiography was performed, resulting in a reliable diagnosis. Moreover, in a patient with isolated occlusion of the P1 segment, as presented in case 3, spatial resolution of MR

or CT angiography may not be sufficient to diagnose such a small lesion. Therefore, combination of vertebral and carotid injections helped to detect isolated P1 occlusion as well as occlusion of the PCA involving the P1 segment and to perform timely interventions. However, combination angiography still has a limitation. Because the absence of a P1 segment with ipsilateral fetal-type origin of a PCA on the vertebral and carotid injections does not prove the absence of bilateral paramedian ischemia due to isolated AOP occlusion,¹³ its possibility should still be investigated using high-magnification vertebral angiography.

Lessons

Using endovascular treatment, 3 patients with bilateral paramedian thalamic stroke due to the occlusion of the unilateral P1 segment were treated successfully. Because timely intervention can dramatically ameliorate the symptoms, awareness of both the variants of bilateral supply of the paramedian thalamic artery from a single P1 segment and the life-threatening presentation of occlusion of such variants is important. A combination of vertebral and carotid injections, rather than MR or CT angiography, is necessary for reliable diagnosis of the small vascular lesions.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Akamatsu, Ogasawara. Acquisition of data: Akamatsu, Yoshida, Kojima, Miyoshi, Kashimura. Analysis and interpretation of data: Akamatsu. Drafting the article: Akamatsu, Yoshida. Critically revising the article: Akamatsu, Yoshida, Ogasawara. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Akamatsu. Study supervision: Ogasawara.

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