Acute basilar artery (BA) occlusion is a devastating form of posterior circulation stroke, with a high risk of long-term disability or death if recanalization is not performed early. Performance of emergent recanalization therapy is determined synthetically based on symptom onset time, stroke severity, and brain imaging. Herein, we report the case of a 72-year-old male patient presenting with minor neurological symptoms, but with thrombotic occlusion at the BA tip. Transcranial Doppler (TCD) showed systolic flow reversal along the vertebrobasilar arteries, and the patient benefited from endovascular thrombectomy. This case highlights the critical role of TCD in identifying hemodynamic insufficiency and determining the implementation of endovascular interventions to mitigate stroke progression.

**CASE**

The patient was a 72-year-old man who was a current 15 pack-year smoker and had been diagnosed with hypertension and dyslipidemia. His medications included candesartan 8 mg, atorvastatin 10 mg, fenofibrate 160 mg, and antiplatelet drugs including cilostazol 100 mg and aspirin 100 mg. He suddenly developed dizziness immediately after going to the bathroom in the middle of the night (onset-to-arrival time, 66 minutes). Upon arrival, his blood pressure was 140/92 mmHg and National Institutes of Health Stroke Scale (NIHSS) score was 0. Two hours and ten minutes after the onset of dizziness, he further developed dysarthria, left facial palsy, left-sided motor weakness, and subjective sensory numbness. Brain diffusion-weighted imaging performed 2 hours and 50 minutes after onset revealed faint high signal intensities in the right peripheral pons, right superior cerebellum, and right middle cerebellar peduncle (Fig. 1A, 1B, and 1C, respectively). His neurological symptoms, including left lower facial palsy and mild dysarthria, were alleviated (NIHSS 2). Brain magnetic resonance angiography (MRA)
nine hours after onset showed occlusion at the BA tip, but the flow patency of both posterior cerebral arteries (PCAs) was preserved (Supplementary Fig. 1A, 1B). Notably, neck MRA revealed no stenosis or occlusion of the subclavian artery proximal to the origin of the vertebral artery (Supplementary Fig. 1C). Furthermore, the aortic arch was normal, and there was no evidence of compression of the subclavian artery (e.g., Takayasu arteritis or cervical rib).

Baseline TCD demonstrated initial systolic flow reversal along the BA and left vertebral artery due to distal BA occlusion (Fig. 2A); however, his neurological symptoms remained unchanged (NIHSS 2). His blood pressure was 132/86 mmHg before TCD. Two days after symptom onset, transfemoral cerebral angiography revealed the presence of a BA tip thrombus that blocked the orifice of the right PCA; however, its distal flow was maintained through the ipsilateral posterior communicating artery (Supplementary Fig. 2A), a finding similar to that of the previous MRA. The thrombus was retrieved in one attempt with suction thrombectomy by an interventional neurologist, and the BA tip was recanalized using TICI (thrombolysis in cerebral infarction) 2b (Supplementary Fig. 2B). His neurological symptoms gradually disappeared, and follow-up TCD one day after thrombectomy showed retrograde systolic flow at the proximal BA with a low-amplitude (weak flow) waveform at the distal BA, suggesting partial improvement in flow (Fig. 2B). Follow-up TCD performed 4 days after the thrombectomy revealed a near-normal flow pattern and direction along the vertebrobasilar track (Fig. 2C). His blood pressure was 137/82 mm Hg prior to the follow-up TCD study. Further serial follow-up TCD could not be performed because of the cost burden. The embolic source of the stroke remained undetermined despite a full cardioembolic workup. Secondary stroke prevention was managed with antiplatelet drugs (clopidogrel 75 mg and aspirin 100 mg for four weeks, followed by clopidogrel 75 mg thereafter) and high-dose statin. Three months later, the patient was neurologically free of symptoms, and follow-up brain MRA showed complete recanalization of the BA tip (Supplementary Fig. 2C); however, T2-weighted imaging revealed focal high intensity in the crus cerebri of the right lower midbrain due to previous BA tip occlusion (Fig. not shown).

DISCUSSION

Subclavian artery steal is asymptomatic in most patients. In patients with flow reversal of the vertebral artery ipsilateral to a subclavian stenosis/occlusion, 76% had antegrade flow in the BA.4 Patients with antegrade flow in the BA are less likely to experience neurological symptoms; however, our patients had minor and stationary neurological symptom despite the retrograde systolic flow in the BA on TCD.

We acknowledge that our patient did not comply with

Fig. 1. Initial diffusion weighted imaging showing faint acute ischemic lesions in the right peripheral pons (A), superior cerebellum (B), and middle cerebellar peduncle (C).
Fig. 2. Subclavian steal on Transcranial Doppler without arterial compression. (A) Initial systolic flow reversal with loss of diastolic flow along the basilar artery (BA) and left vertebral artery. (B) Follow-up study 1 day after thrombectomy showing retrograde systolic flow at the proximal BA with low-amplitude waveform at the distal BA. (C) Follow-up investigation 4 days after thrombectomy showing a near normal flow pattern and direction.
the protocol for acute stroke management, notwithstanding the patient’s visit within the golden time. His neurological symptoms were minor (left facial palsy and dysarthria according to the NIHSS 2), and deciding on endovascular treatment was challenging due to the discrepancy between the initial occlusion at the BA tip and minor neurological symptoms. However, TCD findings of retrograde flow from the BA provided critical information regarding the likelihood of a high risk of neurological deterioration, encouraging prompt endovascular treatment.

In this case, it remains unclear why the retrograde systolic flow along the vertebrobasilar track recovered slowly despite successful recanalization. Notably, TCD after thrombectomy (Fig. 2B) demonstrated a paucity of retrograde flow in the BA at depths of 94 and 100 mm, in contrast to the findings from the BA at depths of 80-90 mm. This phenomenon could be ascribed to the low blood volume reaching the distal BA owing to retrograde flow below the mid-BA level. The waveform in the distal BA showed a blunting pattern, suggesting weak flow. The temporal discrepancy is speculated to be ascribed to remodeling from an invisible residual thrombus between the clinical improvement following reperfusion therapy and the normalized time point of TCD. Nevertheless, the degree of flow reversal showed a sequential decrease, and our patient showed no fluctuations in blood pressure or newly developed neurological symptoms after mechanical thrombectomy.

TCD is a feasible, and valid non-invasive bedside method for evaluating the cerebral hemodynamics in acute stroke setting. The systolic flow reversal along the vertebrobasilar track was an acutely developing finding, which is distinct from largely asymptomatic chronic subclavian steal syndrome. This was a unique case of posterior circulation stroke demonstrating a subclavian-basilar steal phenomenon on real-time TCD without prevertebral subclavian artery stenosis. Given that acute BA occlusion is known to be a high-risk factor for grave stroke and clinicoradiological dissociation as a minor neurological symptom with large vessel occlusion delaying timely treatment, TCD is suggested to play a critical role in identifying hemodynamic insufficiency and determining the implementation of endovascular intervention to mitigate stroke progression.

SUPPLEMENTARY MATERIALS

Supplementary materials related to this article can be found online at https://doi.org/10.31728/jnn.2024.00151

Ethics Statement

Written informed consent was obtained from the patient to report demographic data, medical conditions, neuroimaging, and treatment.

Availability of Data and Material

The imaging data obtained or edited in this study are available from the corresponding author upon request.

Author Contributions

All the authors contributed to the work described in this article. Drs. Park and Yoon conceived of and designed the experiments, and drafted the manuscript. Dr. Choi and Dr. Yoon were involved in the data collection and clinical management of the patient. Dr. Park reviewed, supervised, and edited the manuscript.

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Conflicts of Interest

No potential conflicts of interest relevant to this article was reported.

REFERENCES


Supplementary Fig. 1. TOF-MRA (A and B) and contrast-enhanced neck magnetic resonance angiography (MRA) (C). (A) The basilar artery (BA) tip just before the posterior cerebral artery (PCA), and the right PCA is occluded due to a thrombus. (B) The flow patency of both PCAs is maintained. (C) Neck MRA showing no stenosis or occlusion of the subclavian artery proximal to the origin of the VA. TOF, Time-of-flight; VA, vertebral artery.
Supplementary Fig. 2. Transfemoral cerebral angiography (A and B) and magnetic resonance angiography (MRA) (C) of the posterior circulation. (A) The basilar artery (BA) tip and orifice of the right posterior cerebral artery (PCA) are not visible because of the thrombus, but the distal flow of the PCA is maintained via the ipsilateral posterior communicating artery (arrow). (B) Following one-step suction thrombectomy, complete filling below the BA tip can be visualized; however, the filling is slower than normal (TICI 2b). (C) Follow-up brain MRA showing complete recanalization below the BA tip. TICI, thrombolysis in cerebral infarction.