

Positional cervical Doppler ultrasonography suggesting proximal vertebral artery obstruction in Bow Hunter syndrome: a case report

Taibo Uchida

University of Fukui

Yuki Kitazaki

`turkey@u-fukui.ac.jp`

University of Fukui

Tomohisa Yamaguchi

University of Fukui

Hirotohi Hamaguchi

Kita Harima Medical Center

Noriko Matsumoto

Nippon Medical School

Mayu Eguchi

University of Fukui

Kojiro Usui

University of Fukui

Rei Asano

University of Fukui

Naoko Takaku

University of Fukui

Soichi Enomoto

University of Fukui

Masamichi Ikawa

University of Fukui

Osamu Yamamura

University of Fukui

Yasuhiro Nishiyama

University of Fukui

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Additional Declarations: No competing interests reported.

Abstract

Background

Bow Hunter syndrome results from dynamic vertebral artery stenosis or occlusion during neck rotation.

Case presentation:

A man in his 70s presented with dizziness, nausea, and vomiting and was diagnosed with left occipital and right cerebellar infarctions. On hospital day 7, rightward neck rotation reproduced the dizziness. Positional cervical Doppler ultrasonography showed normal antegrade flow in the left vertebral artery in the neutral position, a to-and-fro waveform immediately after rotation, and partial recovery after approximately 30 s with a delayed systolic peak. These findings suggested an intermittent obstruction proximal to the sampling site. Computed tomography angiography in the neutral and rightward-rotated positions demonstrated kinking and slit-like narrowing of the proximal left vertebral artery at T1. Rotational digital subtraction angiography confirmed marked flow reduction. Computed tomography angiography revealed no fixed extravascular compressive lesions. The patient was treated with aspirin and restriction of neck rotation.

Conclusions

Positional Doppler waveform analysis may help guide the selection of anatomical imaging, although angiographic confirmation remains necessary.

Background

Bow Hunter syndrome (BHS) is a rare cause of posterior circulation ischemia resulting from dynamic stenosis or occlusion of a vertebral artery (VA) during neck rotation [1, 2]. The responsible lesion typically occurs at the atlantoaxial or subaxial cervical levels, whereas proximal V1 involvement is uncommon [2–4].

Since the obstruction is position-dependent, vascular imaging performed in the neutral position may not identify the causative lesion. Digital subtraction angiography (DSA), computed tomography angiography (CTA), magnetic resonance angiography, and Doppler ultrasonography during neck rotation can demonstrate positional changes in VA patency or blood flow [2, 4]. Doppler ultrasonography provides a noninvasive assessment of these hemodynamic changes [5–7]. However, its ability to indicate whether the obstruction is proximal or distal to the sampling site remains uncertain.

Here, we report a case of BHS caused by rotation-induced kinking and stenosis of the proximal left VA at the T1 level. Positional cervical Doppler ultrasonography suggested that the obstruction was proximal to

the sampling site. Positional CTA identified the T1-level kink, and rotational DSA confirmed marked flow reduction.

Case presentation

This case report was prepared in accordance with CAsE REport (CARE) guidelines.

Patient information and initial assessment

A 73-year-old man developed sudden dizziness, nausea, and repeated vomiting while lying supine. No association with neck rotation was observed initially. His medical history included hypertension and glaucoma, managed with azilsartan and amlodipine, respectively. He had a 23-pack-year smoking history. On admission, his blood pressure was 150/93 mmHg, and his heart rate was 85 beats/min in sinus rhythm. Neurological examination revealed a right homonymous hemianopia without nystagmus, ocular motor abnormalities, facial weakness, dysarthria, limb weakness, sensory deficits, or cerebellar ataxia. His National Institutes of Health Stroke Scale score was 2.

Diffusion-weighted magnetic resonance imaging (MRI) showed acute infarctions in the left occipital lobe and right cerebellar hemisphere (Fig. 1a, b). Cervical magnetic resonance angiography (MRA) showed poor and preserved visualization of the right and left VA, respectively (Fig. 1c). Intracranial MRA showed segmental occlusion of the left posterior cerebral artery (PCA) (Fig. 1d). Cervical duplex ultrasonography revealed no significant abnormalities in the common or internal carotid arteries. The right VA had a preserved caliber; however, no Doppler flow signal was detected at its origin. The initial contrast-enhanced CTA showed no false lumen, intimal flaps, or other findings suggestive of VA dissection.

Coagulation studies and hepatic, renal, and thyroid function test results were within the reference ranges. The D-dimer level was 0.9 µg/mL, the hemoglobin A1c level was 5.2%, and the low-density lipoprotein cholesterol level was 88 mg/dL. Electrocardiography and 24-hour Holter monitoring indicated no atrial fibrillation. Transthoracic echocardiography showed no regional wall motion abnormality, left atrial enlargement, or intracardiac thrombus. Dual antiplatelet therapy with aspirin and clopidogrel was initiated for presumed noncardioembolic ischemic stroke.

Diagnostic assessment and clinical course

On hospital day 7, dizziness recurred when the patient was supine and reached for an object with his right arm. During this maneuver, the patient rotated his neck to the right. Further history-taking established that the initial episode had also occurred during rightward neck rotation. Repeat MRI revealed no new infarction. Follow-up MRA showed recanalization of the left PCA, and the left occipital infarction showed hemorrhagic transformation. The reproducible positional trigger prompted evaluation for BHS.

Positional cervical Doppler ultrasonography was performed with the sample volume placed in the mid-cervical V2 segment of the left VA. The examination was performed in the neutral position and during rightward neck rotation. In the neutral position, the left VA showed normal antegrade flow (Fig. 2a). Rightward neck rotation reproduced the dizziness and produced a to-and-fro waveform consistent with severe transient flow limitation (Fig. 2b). Although the rotated position was maintained, antegrade flow partially recovered after approximately 30 s. The recovered waveform showed a delayed systolic peak, consistent with a poststenotic pattern (Fig. 2c). The waveform sequence suggested an intermittent flow-limiting obstruction proximal to the sampling site. The corresponding patient and transducer positions are shown in Fig. 2d and e, respectively.

DSA was performed on hospital day 21. In the neutral position, the left VA showed normal antegrade filling, and the distal right VA was filled retrogradely from the vertebrobasilar junction (Fig. 3a). During rightward neck rotation, antegrade flow in the left VA markedly decreased. Rotational angiography also showed to-and-fro movement of the contrast medium in the proximal left VA. The selected image demonstrates marked flow reduction in the affected segment (Fig. 3b).

A comparison of three-dimensional CTA images obtained in the neutral and rightward-rotated positions showed accentuation of kinking of the proximal left VA during rightward rotation (Fig. 4a–d). A volume-rendered image with the osseous structures retained demonstrated the anatomical relationship between the proximal left VA and the cervicothoracic skeleton (Fig. 4e). The corresponding axial contrast-enhanced source image showed a focal slit-like luminal narrowing of the proximal left VA (arrow, Fig. 4f). Together with the positional Doppler and DSA findings, these CTA findings confirmed that the BHS was caused by rotation-induced kinking and stenosis of the proximal left VA.

Therapeutic intervention and follow-up

Therapeutic intervention and follow-up

Conservative management was selected because no fixed compressive lesion was identified, and restriction of neck rotation controlled the positional symptoms. After short-term dual antiplatelet therapy, clopidogrel was discontinued on hospital day 21. The patient was discharged on hospital day 26 with aspirin 100 mg/day and a cervical collar. No recurrent symptoms occurred during the year following discharge.

Discussion and conclusions

The main observation in this case was that positional cervical Doppler waveform analysis suggested the presence of a flow-limiting lesion proximal to the sampling site prior to anatomical confirmation. Positional CTA subsequently identified rotation-induced kinking of the proximal left VA at the T1 level, and rotational DSA confirmed a marked flow reduction. The agreement among symptom reproduction, Doppler findings, and positional angiographic findings supported the diagnosis.

The locations and mechanisms of these lesions were unusual. BHS generally involves the atlantoaxial or subaxial VA [2–4]. In a review of 126 cases, only 5 (4%) involved the V1 segment [3]. In the present patient, neck rotation accentuated proximal VA kinking without an osteophyte, tumor, or other fixed compressive structure. This mechanism involve dynamic vascular deformation rather than fixed extrinsic compression.

The principal contribution of Doppler ultrasonography is its relative hemodynamic localization. Previous reports have shown that cervical or transcranial Doppler ultrasonography can detect rotation-induced changes in the VA or posterior circulation flow in BHS [5–7]. Duplex ultrasonography can also provide indirect information on the level of VA occlusion [8]. In this case, the to-and-fro waveform and the subsequent partial recovery with a delayed systolic peak were consistent with severe intermittent obstruction proximal to the sampled segment. This interpretation narrowed the target area for subsequent imaging.

Partial flow recovery during sustained rotation was also observed. The V1 segment extends from the subclavian artery to the transverse foramen and may be more mobile than the intraforaminal VA [9]. Sustained rotation may have altered the geometry of the proximal VA, partially releasing the kink or permitting elastic recoil.

The infarct pattern was compatible with both the hemodynamic and embolic mechanisms. The absence of an antegrade signal at the right VA origin and the retrograde filling of its distal segment suggest a limited antegrade contribution from the right VA. Therefore, the posterior circulation may have depended substantially on the left VA, and the rotation-induced reduction of left VA flow may have contributed to the right cerebellar infarction through transient hypoperfusion [10]. In contrast, the initial left PCA occlusion, subsequent recanalization, and hemorrhagic transformation of the left occipital infarction were compatible with, but not specific for, reperfusion after embolic occlusion. Transient proximal VA kinking may have promoted local thrombus formation and artery-to-artery embolism, as reported in Bow Hunter stroke [11].

Conservative treatment was selected because no fixed compressive lesion was present and the provoking movement could be restricted. Favorable outcomes with conservative management have been reported in selected patients with rotational VA occlusion, and fusion-based procedures have also been used to prevent recurrent positional occlusion [10, 12]. Rotation-induced kinking of the left proximal VA at the T1 level is an uncommon cause of BHS. In this patient, positional cervical Doppler waveform analysis indicated that the obstruction was proximal to the sampling site, and guided targeted vascular imaging. Treatment should be individualized according to the anatomical cause, recurrence risk, suspected ischemic mechanism, and feasibility of avoiding the provoking movement.

Declarations

Ethics approval and consent to participate

This case report was conducted in accordance with the Declaration of Helsinki and local institutional requirements. The Institutional Review Board of the University of Fukui waived the requirement for written informed consent to participate because of the retrospective nature of the report, and an opt-out procedure was used.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Competing interests

The authors declare that they have no competing interests.

Authors' information

Not applicable.

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Author Contribution

TU: conception, interpretation of this case, and writing the manuscript. YK: revising the manuscript and critical review of the manuscript. TY: conception, interpretation of this case and revising the manuscript. HH, NM: interpretation of the imaging results, and critical review of the manuscript. MY, KU, RA, NT and SE: conception, and revising the manuscript. MI, OY: conception, interpretation of this case. YN: conception, interpretation of this case, and critical review of the manuscript. All authors contributed to the study conception, read and approved the final manuscript.

Data Availability

Availability of data and materials All data generated or analyzed during this study are included in this article and its additional files. Additional clinical information is available from the corresponding author on reasonable requests, subject to patient privacy and institutional requirements.

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Figures

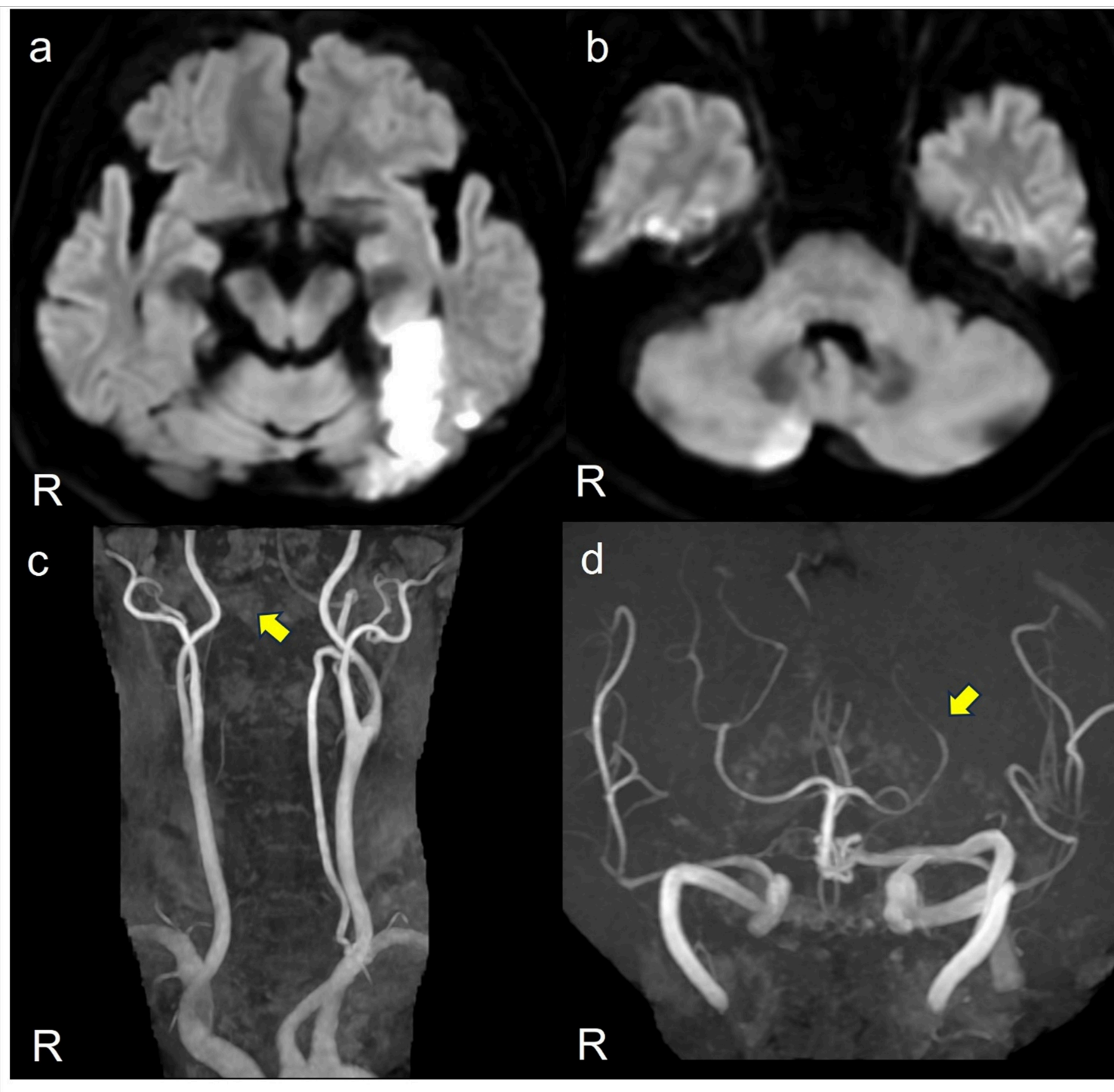


Figure 1

Initial magnetic resonance imaging and angiography findings. (a) Diffusion-weighted imaging shows an acute infarction in the left occipital lobe. (b) Diffusion-weighted imaging shows an acute infarction in the right cerebellar hemisphere. (c) Cervical magnetic resonance angiography shows poor visualization of the right vertebral artery (arrow) and preserved visualization of the left vertebral artery. (d) Intracranial magnetic resonance angiography shows segmental occlusion of the left posterior cerebral artery (arrow).

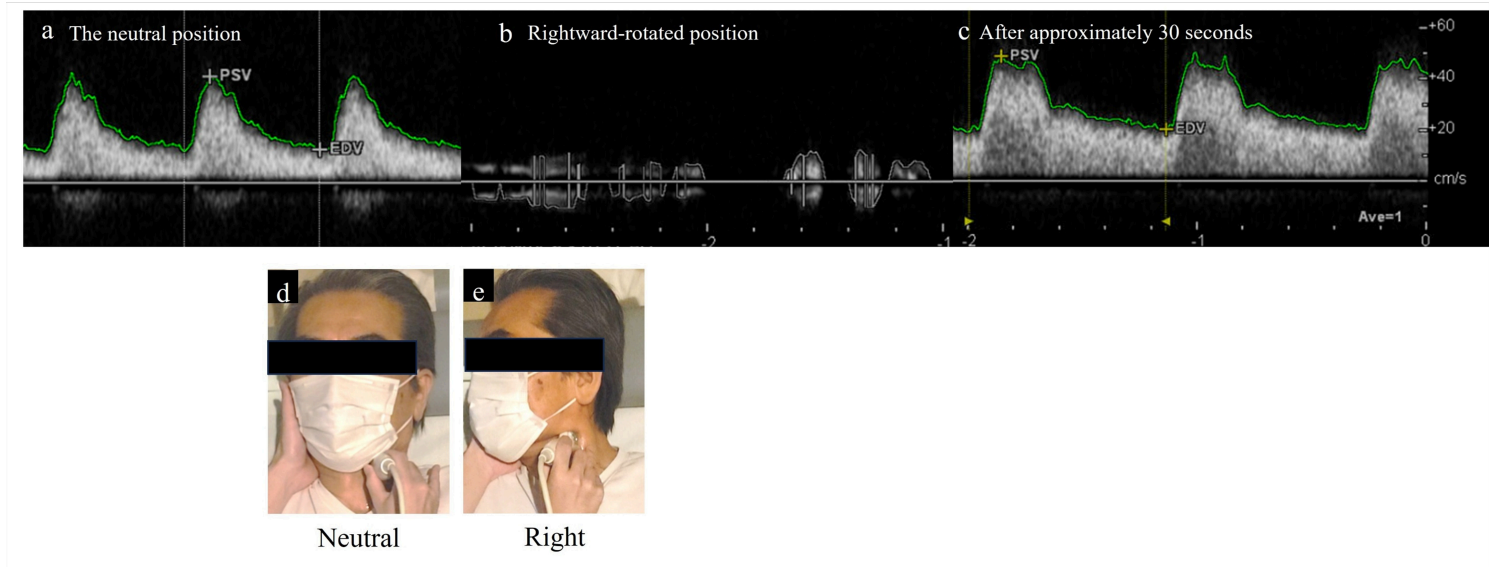


Figure 2

Positional Doppler waveforms of the left vertebral artery. The sample volume was placed in the mid-cervical V2 segment of the left vertebral artery, and spectral Doppler waveforms were recorded in the neutral position and during rightward neck rotation. (a) Normal antegrade flow in the neutral position. (b) Immediately after rightward neck rotation, dizziness recurred, and a to-and-fro waveform was observed, consistent with severe transient flow limitation. (c) After approximately 30 s of sustained rightward neck rotation, antegrade flow partially recovered, and the waveform demonstrated a delayed systolic peak consistent with a poststenotic pattern. (d) Patient and transducer position during waveform acquisition in the neutral position. (e) Patient and transducer position during waveform acquisition with rightward neck rotation.

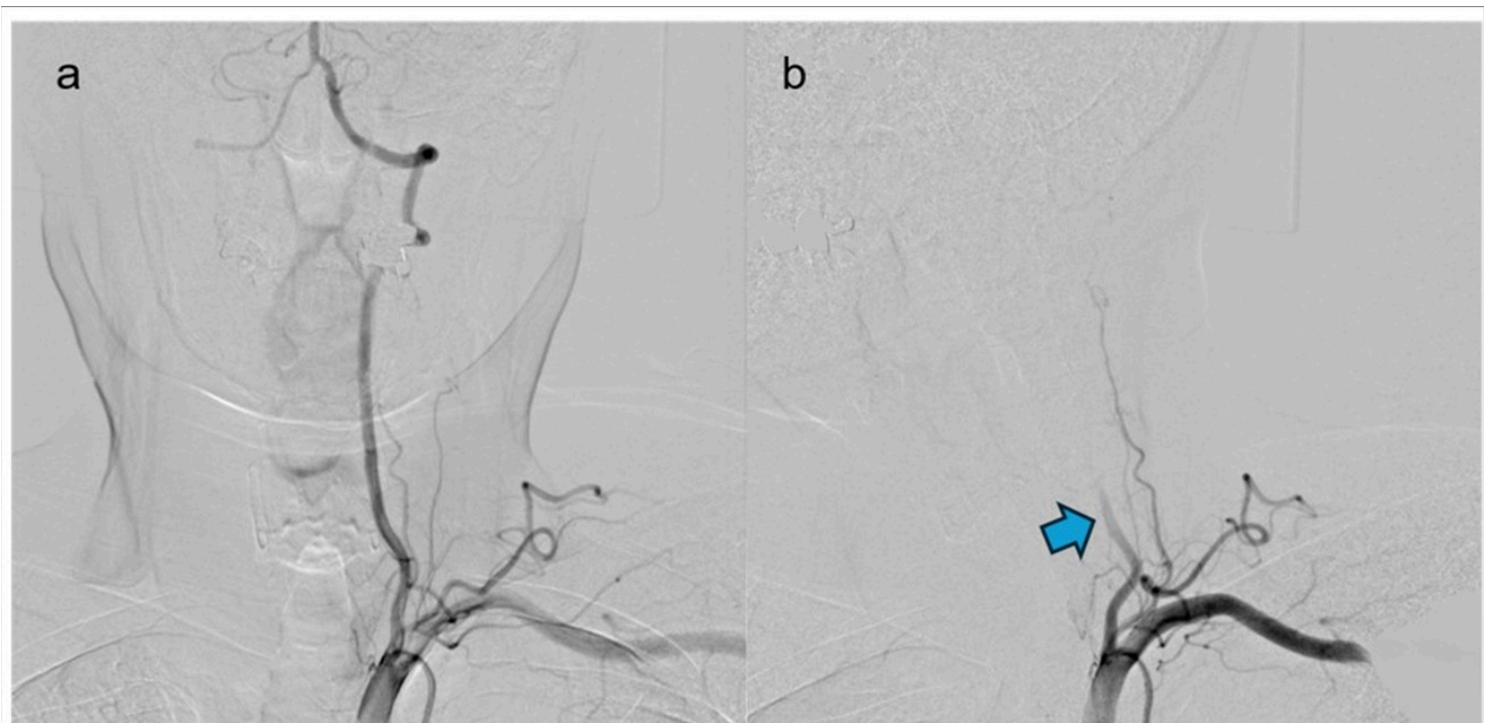


Figure 3

Positional Doppler waveforms of the left vertebral artery. The sample volume was placed in the mid-cervical V2 segment of the left vertebral artery, and spectral Doppler waveforms were recorded in the neutral position and during rightward neck rotation. (a) Normal antegrade flow in the neutral position. (b) Immediately after rightward neck rotation, dizziness recurred, and a to-and-fro waveform was observed, consistent with severe transient flow limitation. (c) After approximately 30 s of sustained rightward neck rotation, antegrade flow partially recovered, and the waveform demonstrated a delayed systolic peak consistent with a poststenotic pattern. (d) Patient and transducer position during waveform acquisition in the neutral position. (e) Patient and transducer position during waveform acquisition with rightward neck rotation.

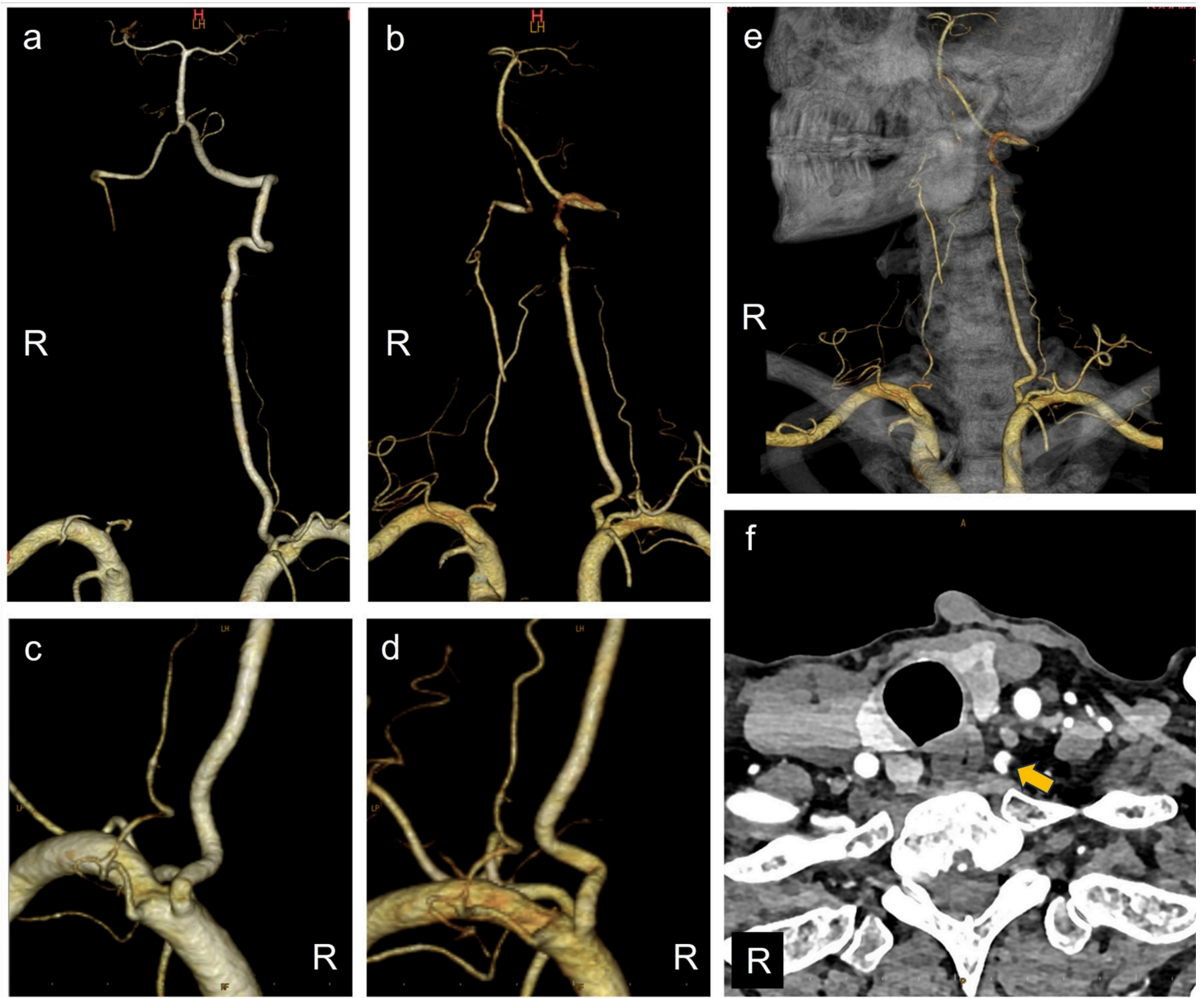


Figure 4

Rotational digital subtraction angiography of the left vertebral artery. (a) In the neutral position, a frontal projection showed normal antegrade opacification of the left vertebral artery. (b) During rightward neck rotation in the same projection, antegrade opacification markedly decreased in the proximal left vertebral artery. The arrow indicates the affected proximal segment.