Case study

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Innominate artery occlusion: a case study

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Keywords
CT; brachiocephalic artery occlusion; innominate artery occlusion; subclavian steal syndrome; carotid ultrasound

Abstract

Aim of the study: The aim of this case report is to evaluate carotid duplex and hemodynamic patterns in an asymptomatic male patient with innominate artery occlusion. Innominate artery occlusion is a rare clinical entity that can lead to a range of cerebrovascular symptoms, including arm claudication, subclavian steal syndrome, and stroke. The case report emphasizes key learning points in diagnosing innominate artery occlusion using imaging and physiological methods. **Case description:** A 64-year-old asymptomatic male patient with a history of carotid bruit, hypertension, coronary artery bypass grafting, aortic aneurysm, hyperlipidemia, mild aortic stenosis, long-term tobacco use, and a body mass index of 24 was referred for a carotid ultrasound. **Conclusions:** Innominate artery occlusion is a rare condition requiring a comprehensive assessment of collateralization before any intervention is attempted. Considering waveform features such as transient end-diastolic flow reversal and tardus parvus, along with brachial pressures and transcranial Doppler, can assist in evaluating the extent of disease.

Introduction

Occlusive lesions in the innominate artery are relatively rare clinical entities that can lead to a wide range of cerebrovascular symptoms such as arm claudication, subclavian steal syndrome, and stroke[1–3]. Innominate artery occlusions can result in the diversion of blood flow, leading to the siphoning of blood from the contralateral vertebral artery into the subclavian artery and decreased blood flow in the right common carotid artery (RCCA)[1–5]. Marginal cerebral circulation as a result of compensatory blood flow diversion can lead to ischemic episodes, particularly when exercise stress is afflicted on the right arm[1,5,6].

While innominate artery occlusions typically do not result in symptoms of cerebral dysfunction, unless there are numerous atherosclerotic lesions in cerebral arteries or subpar collateral circulation[1,4,5], they can still pose a risk for arterial insufficiency, which can impact the brain and lead to transient brain ischemia[7]. Although the true incidence of innominate artery occlusion or stenosis remains unknown due to its rare clinical presentation[6], there have been a few case studies describing this uncommon occurrence. Most of these studies have focused on therapeutic approaches[8]. In a previously reported study, distinct hemodynamic changes in the right vertebral and carotid arteries were found in 12 patients. Sonographic abnormalities from this study included reversed or bidirectional flow in the right vertebral artery, mid-systolic deceleration in right carotid system branches, and an elevated left common carotid artery (LCCA)/RCCA ratio[5]. Due to there being no standardized guidelines for the treatment and management of innominate artery occlusion or stenosis[7,8], it is crucial to conduct further research into potential diagnostic and therapeutic interventions to effectively manage innominate artery occlusions and the associated cerebrovascular symptoms[4].

This case study describes a 64-year-old asymptomatic male presenting with a carotid bruit and hypertension. The purpose of this case study is to assess carotid duplex findings and hemodynamic patterns in an asymptomatic patient with innominate artery occlusion.

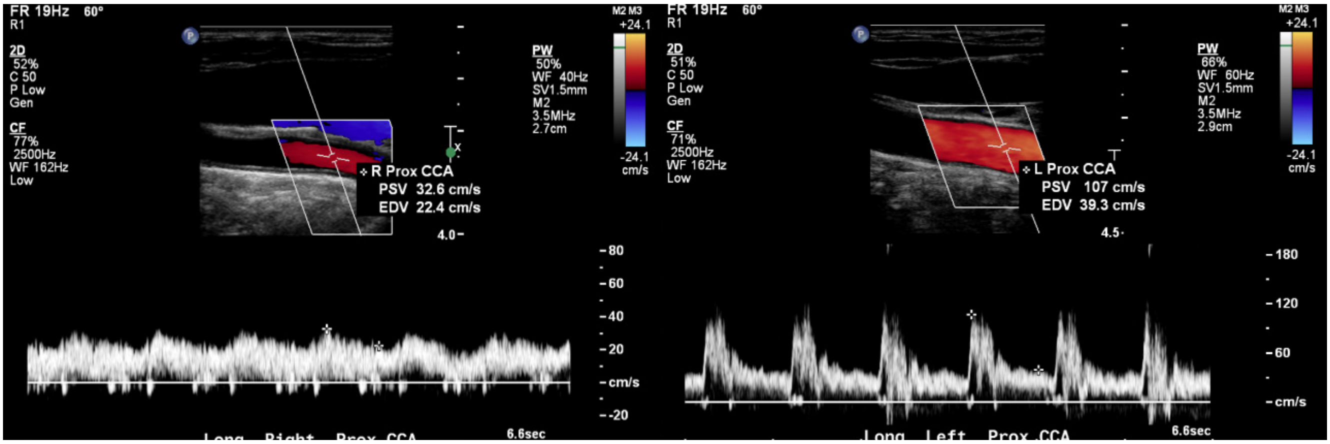

Fig. 1. *Comparison between the dampened waveform in the proximal RCCA and the normal waveform of the proximal LCCA*

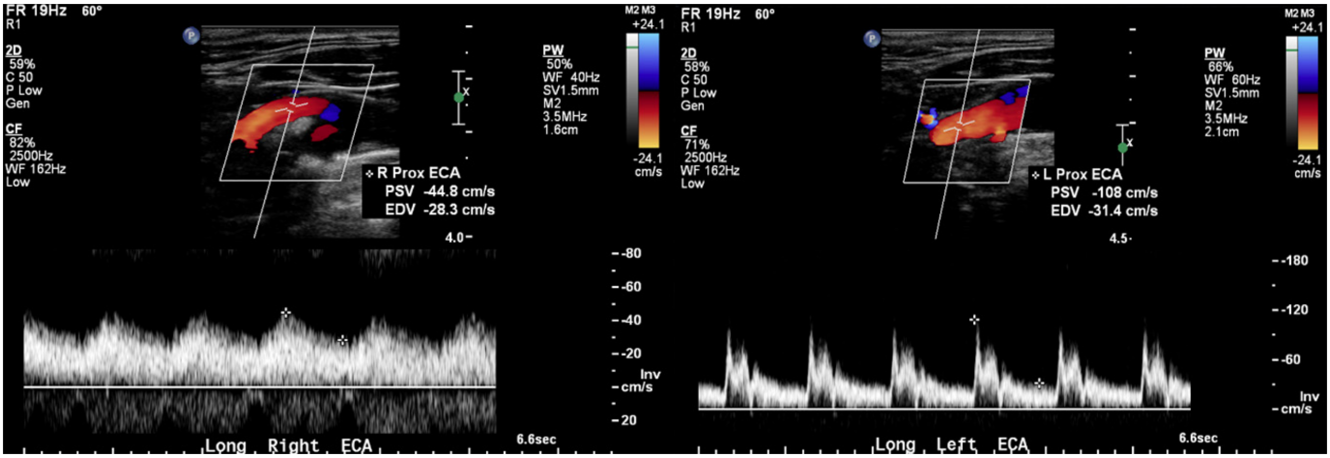

Fig. 2. *Dampened right external carotid artery (RECA) waveform with a PSV of 45 cm/s and end-diastolic velocity (EDV) of 28 cm/s compared to the high-resistance left external carotid artery (LECA) waveform*

Case description

A 64-year-old asymptomatic male patient with a history of left carotid bruit and hypertension was referred for a carotid ultrasound. The patient's medical history also included abdominal aortic aneurysm, chronic obstructive pulmonary disease, coronary artery bypass graft, hyperlipidemia, mild aortic stenosis, and tobacco use. The patient had been smoking approximately a pack of cigarettes every day for 35+ years, and had a BMI of 24.

Ultrasound assessment

On January 6th, 2020, the patient had an ultrasound done in the diagnostic imaging (DI) department. Images were acquired using the Philips IU 22 machine and a L9-3 linear transducer on vascular carotids preset. Once consent was obtained, the patient was placed in the supine position with his neck extended back. Ultrasound findings (Fig. 1, Fig. 2, Fig. 3, Fig. 4) include right mild to moderate atherosclerosis at the carotid bulb, extending into the proximal right internal carotid artery (RICA). The peak systolic velocity (PSV) of the RICA does not exceed 23 cm/s in all segments, while the PSV of the RCCA is 33 cm/s (Fig. 3). The right vertebral artery demonstrates retrograde flow as seen in Fig. 4.

On the left, there is mild to moderate atherosclerosis in the carotid bulb, extending into the proximal ICA. Antegrade flow is demonstrated in the left vertebral artery.

The ultrasound report concludes with the following findings:
- decreased peak systolic velocities of the RICA and LCCA;
- retrograde flow in the right vertebral artery indicating a subclavian steal phenomenon;
- a dedicated computed tomography (CT) angiogram of the neck and circle of Willis is recommended.

CT assessment

Axial thin sections with multiplanar reconstruction were obtained from the aortic arch up through the circle of Willis post intravenous contrast injection on February 7th, 2020. CT findings (Fig. 5, Fig. 6, Fig. 7) include calcified atherosclerotic plaque at the origin of the RICA causing a 30% diameter stenosis.

There is a short segment moderate stenosis where the A1 segment of the right anterior cerebral artery originates. No other intracranial arterial stenosis is identified. The left vertebral artery is dominant relative to the right vertebral artery, with no evidence of stenosis in

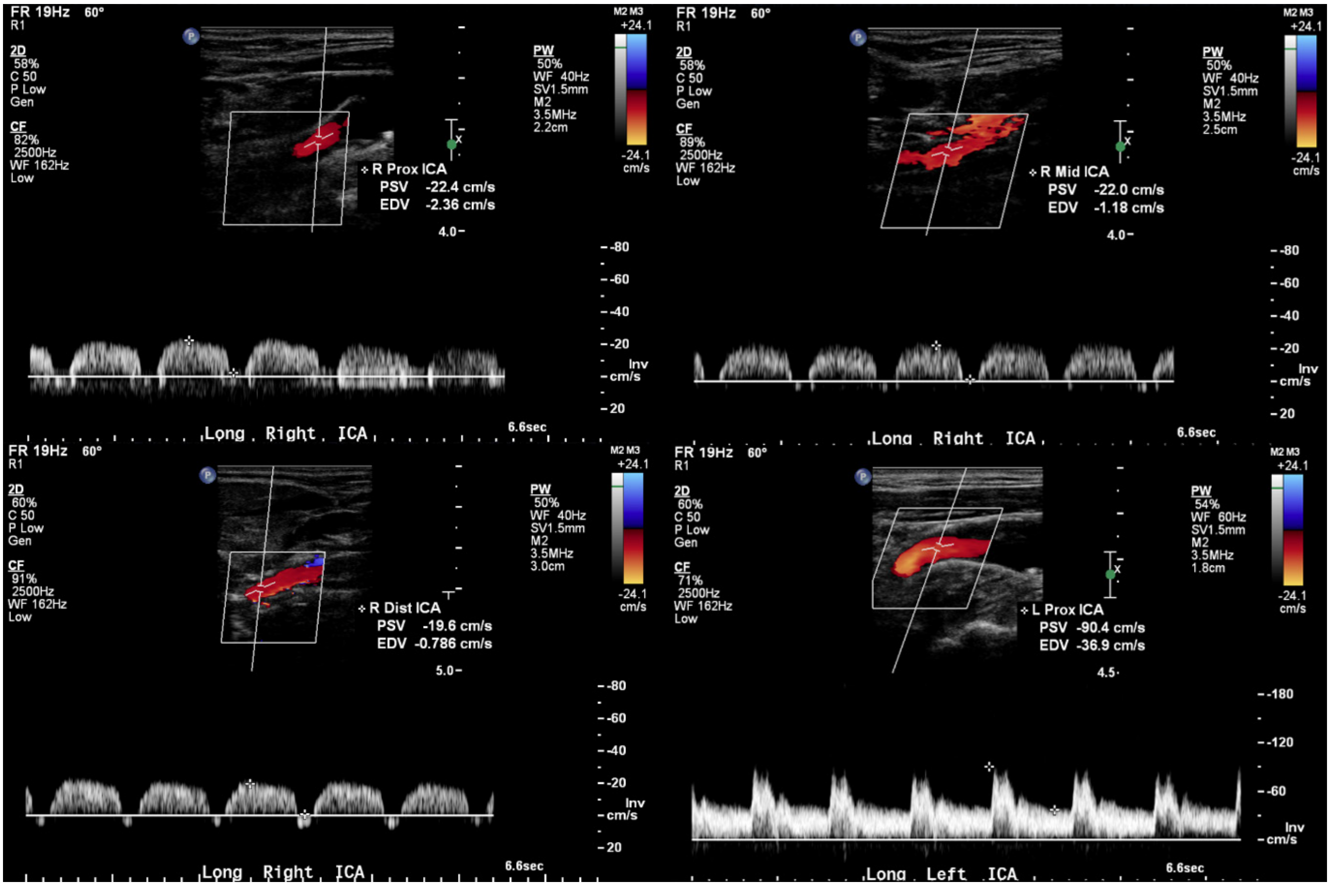

Fig. 3. *Evidence of transient end-diastolic flow reversal and dampening of the waveforms in the distal RICA. The PSV in the RICA does not exceed 23 cm/s in all segments. The proximal left internal carotid artery (LICA) waveform was added for comparison. When imaging the distal ICA, it is important to visualize the parotid gland and to follow the distal ICA, as it dives posteriorly. Increasing the color gain and decreasing the color pulse repetition frequency (PRF) may help visualize the distal ICA*

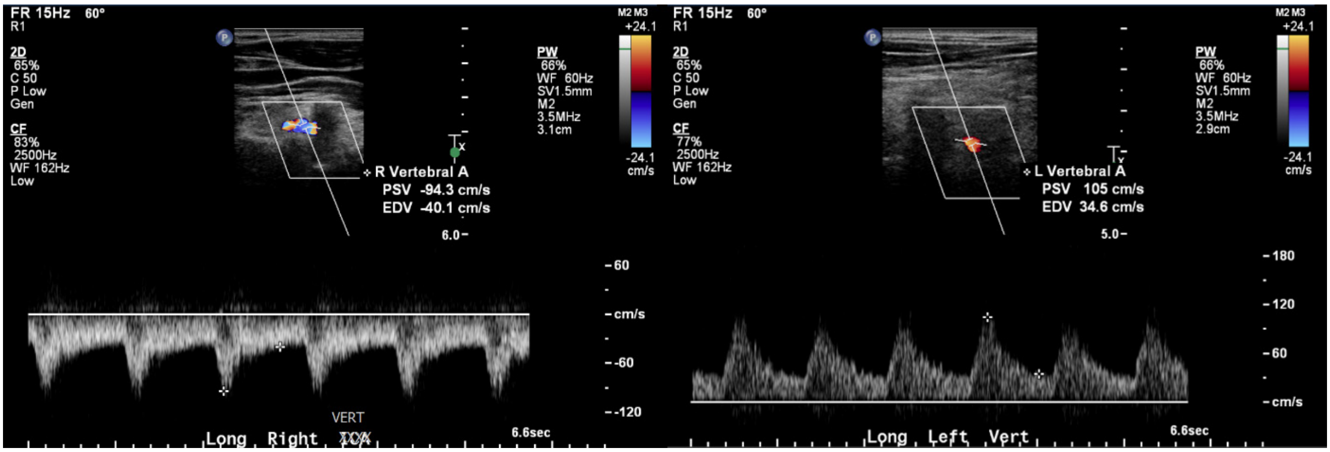

Fig. 4. *Retrograde flow noted in the right vertebral artery with a PSV of 94 cm/s and EDV of 40 cm/s. The left vertebral artery demonstrates antegrade flow with a PSV of 105 cm/s and EDV of 35 cm/s. When the direction of flow is not clear in the vertebral artery, it is advised to document the CCA in split screen, with red representing blood flow towards the brain, and then angling and sliding the probe laterally and posteriorly to image the vertebral artery without changing any of the Doppler parameters. The vertebral vein will be seen anterior to the vertebral artery and may appear pulsatile. It is important not to confuse the two vessels. Both will pass through the transverse foramina of the cervical spine, and it may be easier to distinguish the two by looking for the origin of the vertebral artery off the subclavian artery*

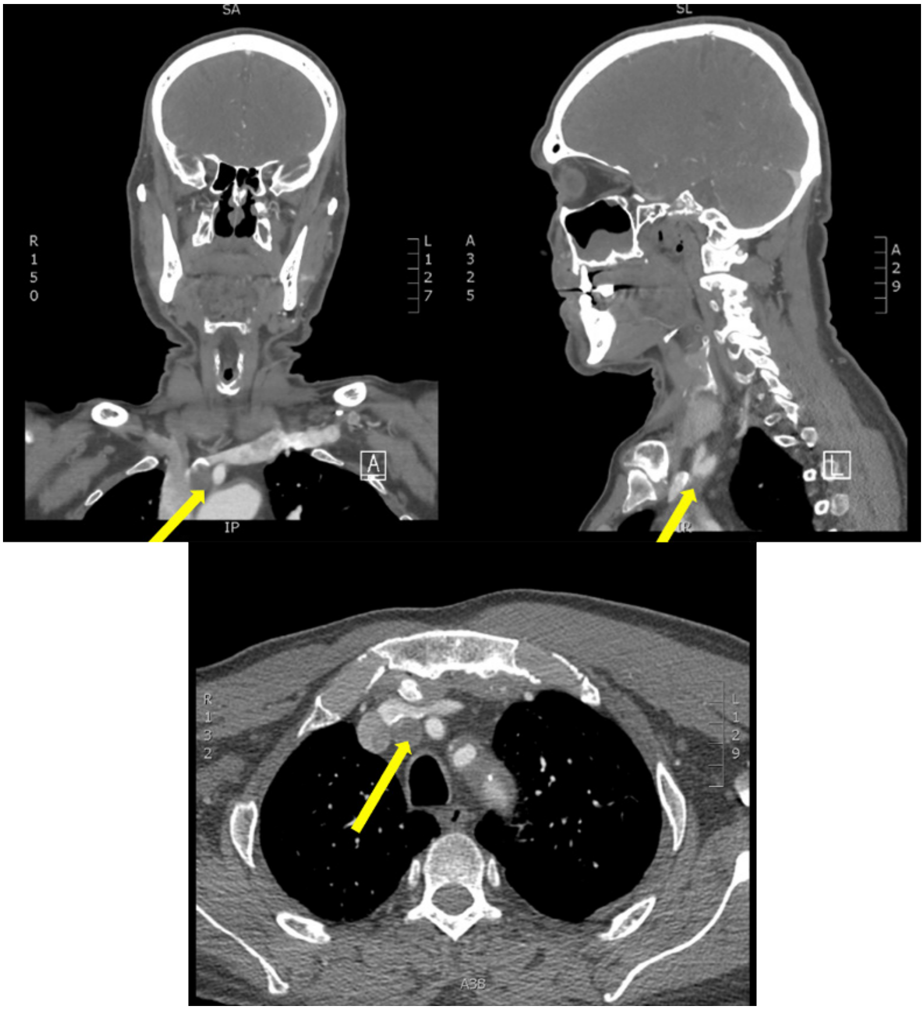

Fig. 5. *Occlusion of the innominate artery is demonstrated proximal to the origin of the RCCA and subclavian artery. Lack of enhancement in the innominate artery is demonstrated in all three planes*

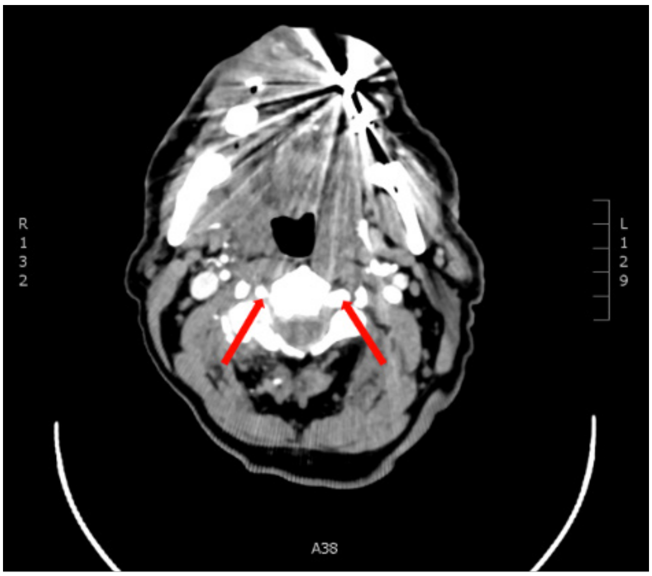

Fig. 6. *Evident left vertebral dominance alluding to contralateral compensatory flow*

either vertebral artery (Fig. 6). Note is made of a moderate stenosis in the supraclinoid segment of the RICA (Fig. 7). The brachiocephalic artery is occluded from the origin to the junction of the right subclavian and RCCA. This finding would explain retrograde flow in the right vertebral artery. The origins of the LCCA and left subclavian arteries are patent and not diseased.

Main report findings include:

- occlusion of the innominate artery;
- 30% lumen diameter reduction at the origin of the RICA;
- moderate stenosis visualized in the supraclinoid RICA.

Non-invasive vascular lab ultrasound assessment

The following year, on September 15th, 2021, the patient was referred to the vascular lab for a carotid ultrasound and lower extremity arterial assessment done by a registered vascular technologist. Ultrasound images were acquired using the Philips IU 22 machine and a L9-3 linear transducer on vascular carotids preset. Physiological testing was done using the Viasonix Falcon diagnostic machine and an 8 MHz continuous-wave (CW) probe. Once verbal consent was

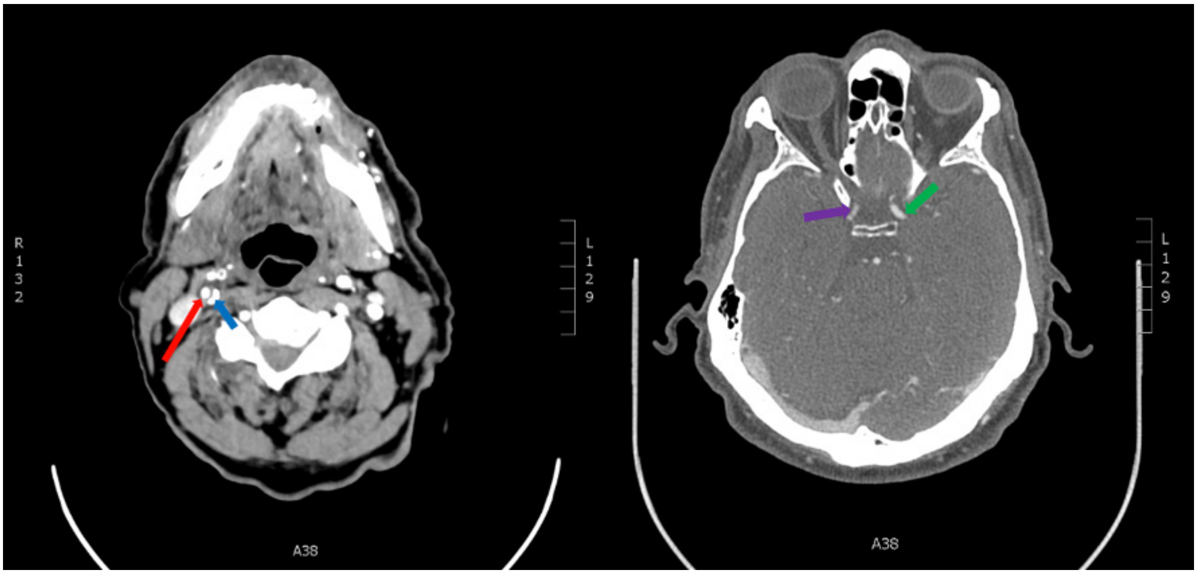

Fig. 7. *Moderate stenosis of the supraclinoid RICA is illustrated by the purple arrow, and calcified plaque (blue arrow) is demonstrated at the origin of the RICA (red arrow), causing a 30% diameter lumen reduction*

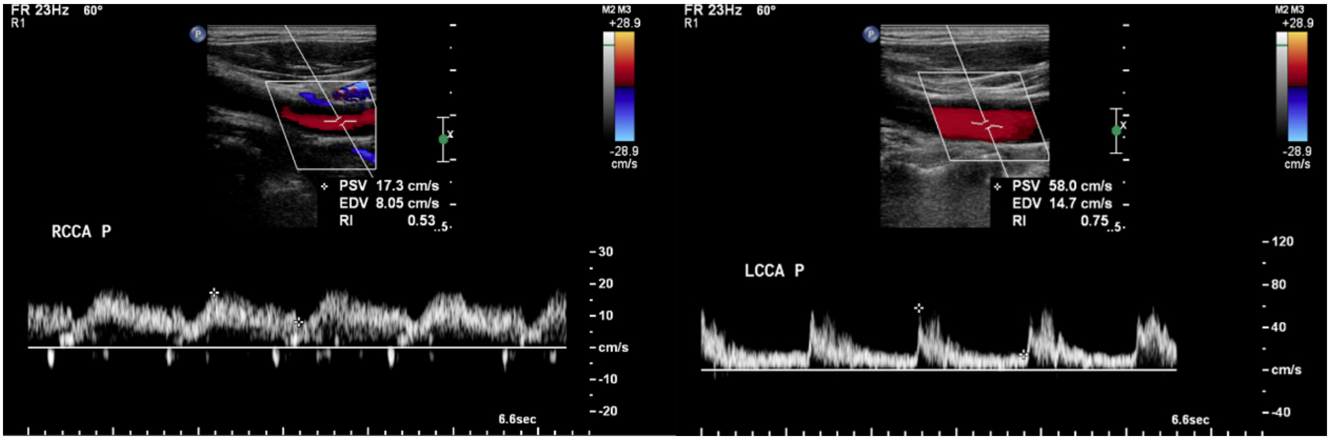

Fig. 8. *Tardus parvus waveform seen in the proximal RCCA*

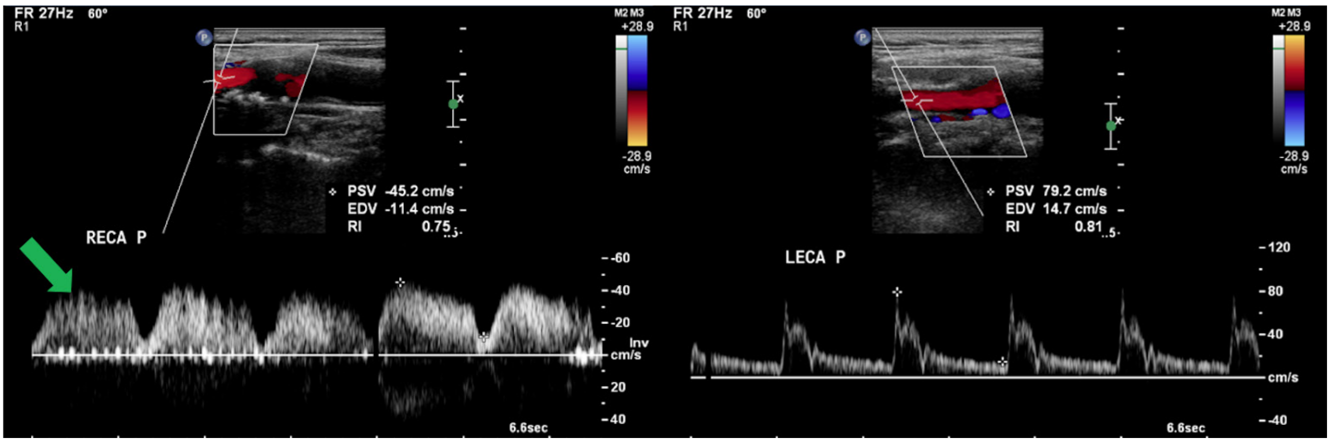

Fig. 9. *Rapid systolic upstroke with a sharp deceleration and diminished forward flow in diastole is characteristic of flow in the LECA. This is in contrast to the prolonged systolic acceleration and rounding of the systolic peak demonstrated in the RECA, consistent with a tardus parvus waveform. Temporal tap was applied on the right side to help distinguish between the RICA and RECA. Reflected waves indicated by the green arrow demonstrate a 'saw-tooth' pattern confirming the identification of the ECA*

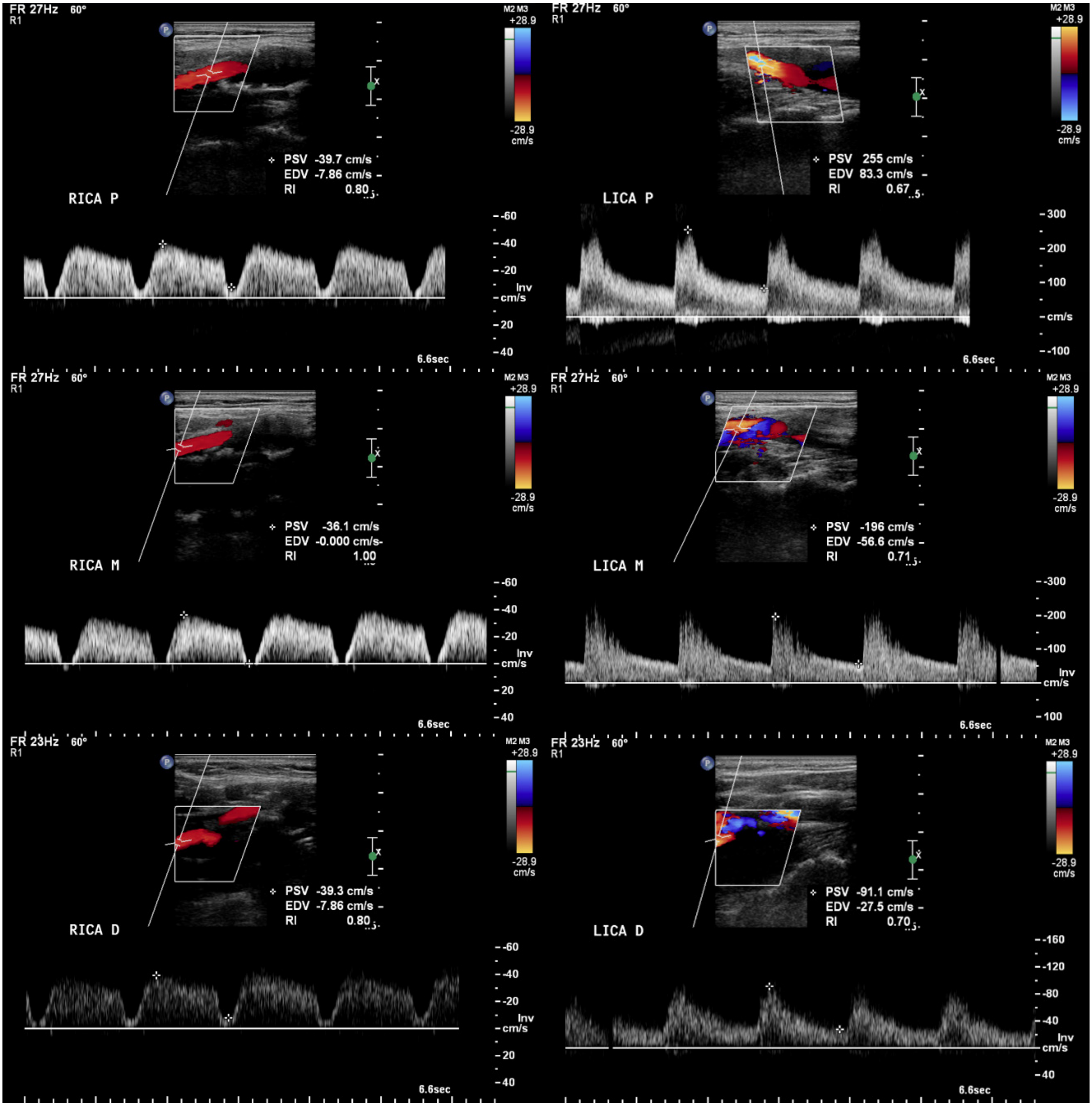

Fig. 10. *Tardus parvus waveform seen in the proximal RICA with spectral broadening. Diminished diastolic flow is seen in all segments of the RICA, signaling a change to high-resistance flow compared to the tardus parvus waveforms visualized in the RECA and RCCA. This may be caused by a moderate stenosis in the intracranial RICA. In the proximal LICA there is a PSV of 255 cm/s indicative of a 50–69% stenosis.*

acquired, the patient was placed in the supine position and asked to relax. The patient was found to have a right brachial pressure of 107 mmHg and a left brachial pressure of 160 mmHg (i.e., a difference of 53 mmHg between the arms). The brachial artery waveform on the right was moderately abnormal. Carotid duplex showed low-resistance monophasic waveforms with prolonged systolic upstroke indicative of a tardus parvus waveform in the RCCA, RECA, and RICA (Fig. 8, Fig. 9), consistent with previous findings. Low velocities in these vessels on the right are indicative of proximal arterial obstruction. In this case, as the CT demonstrated, the proximal obstruction was present in the innominate artery. A mild amount of plaque was found in the right carotid bulb and the proximal RICA, with no significant stenosis. Figure 10 illustrates that the LICA had a mild-moderate amount of echolucent plaque with a PSV of 255 cm/s and LICA/LCCA ratio of 3.92, consistent with a 50–69% stenosis. The right vertebral artery demonstrated a complete subclavian steal phenomenon with compensatory flow contralaterally in the left vertebral artery, with a PSV of 144 cm/s. Additionally, the segmental blood pressures, doppler, and PVR of the patient are shown in Fig. 11.

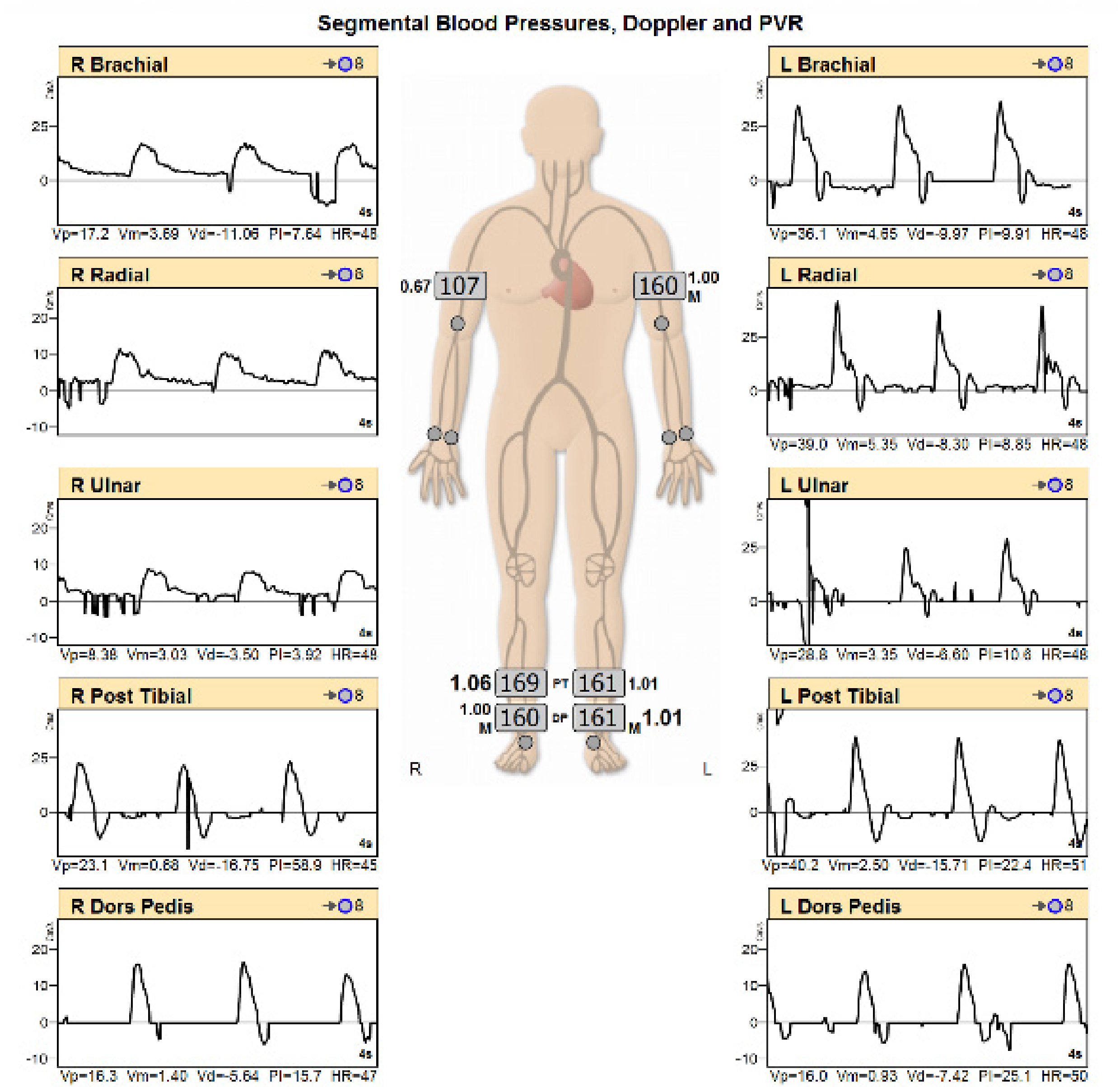

Fig. 11. *Right brachial, radial and ulnar artery waveforms display a rounded peak with a pronounced decrease in amplitude compared to the triphasic left upper extremity waveforms. The difference in brachial pressures across the arms, along with the CW Doppler findings, suggest moderate disease in the right arm. Ankle-brachial indices are in the normal range bilaterally, with multiphasic CW Doppler waveforms.*

The main report findings include:

- complete subclavian steal phenomenon secondary to innominate artery occlusion;
- 50–69% stenosis in LICA with a LICA/LCCA ratio of 3.92;
- drop of 53 mmHg in the brachial pressure in the right arm.

Discussion

Our main finding was the presence of right subclavian steal phenomenon with compensatory contralateral flow in the left vertebral artery. Left vertebral dominance is one of the most common types of collateral circulation, arising as a result of right proximal innominate artery occlusion which decreases blood flow in the right vertebral artery so that, as a compensatory measure, the left side supplies for the lack of flow. In our case, there was no further prominent inflow noted via the collateral circulation from the contralateral ECA or ICA (Fig. 6), nor was there evidence to suggest additional collateral circulation at the time of CT imaging. Retrograde flow into the right vertebral artery indicated a subclavian steal phenomenon.

Subclavian steal phenomenon causes retrograde flow in a vertebral artery as a result of stenosis or occlusion of the subclavian artery before it gives off the vertebral artery. This finding is similar to past case reports[3,6,9], where subclavian steal phenomenon was found resulting from innominate artery occlusion. Şeker *et al.* (2016) reported the case of a 50-year-old male with complaints of repeated syncope episodes, dizziness when tilting his head back, and numbness in the right upper arm while working[3]. His blood pressure in the left arm was 130/70 mm Hg and in the right arm 85/50 mmHg. The symptoms were diagnosed as subclavian steal phenomenon. Similarly, Esen *et al*. (2016) presented a case of double steal phenomenon in a 50-year-old male patient who presented with recurrent syncopal episodes and cerebrovascular ischemia[9]. In both cases, endovascular stenting was performed to restore normal antegrade flow in the common carotid and vertebral arteries, with vascular bypass surgery remaining as an alternative treatment option[9].

Occlusion of the innominate artery, which may be due to atherosclerosis, embolism, trauma, radiation therapy, infections, and congenital abnormalities, may result in significant hemodynamic changes that present with clinical manifestations. As the innominate artery supplies blood to the head, neck, and right upper extremity of the body, common cerebrovascular symptoms can include visual disturbances, upper extremity claudication, head, neck, and arm ischemia, transient ischemic attack, ataxia, and dizziness[10–14]. Compensatory flow mechanisms manifest secondary to an occlusion, showing the intrinsic adaptability of the cerebrovascular system and the drastic pressure gradient changes it can undergo[15].

The findings of this case study emphasize the importance of comprehensive brachial pressure assessment in the diagnosis and management of the condition. Firstly, this case highlights the significance of taking brachial pressure measurements on both arms, as opposed to just one arm, which is a common practice among many practitioners. By only measuring the pressure on the left arm, practitioners may overlook stenosis or occlusion on the contralateral side, leading to a missed diagnosis. Therefore, it is essential to obtain brachial pressure from both arms to ensure accurate assessment and prevent any potential oversight. Systolic pressure readings obtained from each arm play a crucial role in evaluating the severity of the condition.

It is also important to consider possible limitations of ultrasound imaging. Unlike CT, ultrasound is incapable of assessing deep vascular structures beneath the clavicle. Additionally, inter-operator variability is more prevalent with ultrasound than it is with CT[15]. A tardus parvus waveform is composed of a delayed ("tardus") systolic upstroke and a reduced ("parvus") systolic amplitude generating a rounded systolic peak seen distal to a stenosis or, in this case, reconstituted arteries distal to an occlusion[15]. To confidently identify tardus parvus waveforms, it is important to consider whether there is rounding of the systolic peak and dampening of the waveform[12–15]. It is not necessary to identify the source of stenosis or occlusion to confidently identify tardus parvus waveforms, as reflected by this case study[12]. In cases where the disease is limited to one side, making spectral waveform comparisons with the other side may assist in recognizing tardus parvus waveforms.

Furthermore, the number of studies, let alone case studies, reporting innominate artery stenosis findings are meager, as it is a rare phenomenon. Radiographic findings of innominate artery occlusions comprise only 2.5% to 4% of atherosclerotic lesions of the extracranial cerebral arteries, including low grade and asymptomatic stenosis[16]. Consequently, the lack of significant findings regarding the incidence, development, and treatment of innominate artery stenosis limits the clinical application of our study. To add on, due to the nature of case reports, our small sample size further compromises the generalizability, applicability, and validity of the results of this study. It is plausible that other patients may present with similar findings but have different comorbidities or underlying conditions.

Conclusion

As significant complications may arise in patients with innominate artery occlusion, further research should be conducted in order to develop standardized preventive or curative treatments. The role of transcranial Doppler may also be considered in future studies to investigate the extent of disease. In addition, hemodynamic flow in the ophthalmic artery should be considered and evaluated to develop a comprehensive idea of the extent of collateralization.

Conflict of interest

The authors do not report any financial or personal connections with other persons or organizations which might negatively affect the contents of this publication and/or claim authorship rights to this publication.

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Author contributions

Original concept of study: MN, HS. Writing of manuscript: MN, HS, YS, RA, YR, JH, KN. Analysis and interpretation of data: MN, HS, YS, RA, YR, JH, KN. Collection, recording and/or compilation of data: MN, HS, YS, RA, YR, JH, KN. Critical review of manuscript: MN, HS.

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