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Single Case – Cerebrovascular Diseases and Stroke

Carotid Web as a Treatable Etiology of Recurrent Cryptogenic Ischemic Stroke Highlighting Neurointervention: A Case Report and Literature Review

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Keywords
Carotid web · Cryptogenic stroke · Transient ischemic attack · Carotid artery stenting · Case report

Abstract
Introduction: Carotid web is a rare focal variant of intimal fibromuscular dysplasia characterized by a thin, shelf-like projection from the posterior wall of the carotid bulb into the arterial lumen. This abnormality can disturb blood flow and promote thrombus formation, increasing the risk of transient ischemic attacks (TIAs) and ischemic strokes. ***Case Presentation:*** A 55-year-old male, known case of hypertension, presented with sudden-onset dysarthria and left-sided weakness. Initial imaging revealed no acute infarction, and an underlying cause remained unclear. Despite receiving dual antiplatelet therapy and statin, he experienced a recurrent TIA within a week. Digital subtraction angiography revealed a focal shelf-like intraluminal projection in the right internal carotid artery, consistent with a carotid web. Carotid artery stenting (CAS) was performed successfully without any immediate complications. The patient was then commenced on clopidogrel for 3 months and lifelong aspirin. At the 1-month and 3-month follow-ups, he remained symptom-free and had no recurrence. ***Conclusion:*** This case highlights carotid web as a potential but underrecognized cause of recurrent ischemic events, illustrating both the diagnostic challenge of distinguishing it from atherosclerotic plaque with shelf-like configuration and the value of revascularization therapy. Early recognition and timely management by either CAS or carotid endarterectomy can prevent recurrent neurological deficits. Additional studies and case reports are needed to improve understanding of carotid web and to guide the development of evidence-based management strategies.

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Introduction

The carotid web represents a rare intimal variant of fibromuscular dysplasia involving the inner layer of the internal carotid artery. It results from non-atherosclerotic intimal fibrosis and hyperplastic changes, forming a thin, shelf-like ridge arising from the posterior wall of the carotid bulb or the proximal segment of the internal carotid artery. This structure can cause turbulent blood flow and thrombus development, consequently increasing the risk of transient ischemic attack (TIA) or ischemic strokes [1].

Carotid webs are found in approximately 1%–2% of patients with ischemic stroke. However, they are more common in younger patients who suffer from strokes without a clear cause (cryptogenic strokes), with reported prevalence rates as high as 20%–25% [2, 3].

We present the case of a patient with a carotid web who experienced recurrent TIAs and was successfully treated with carotid artery stenting (CAS). This case highlights the diagnostic challenge in identifying the culprit lesion, including the need to distinguish carotid web from focal atherosclerotic plaque with shelf-like configuration. The patient was successfully treated with CAS and has had no recurrence of stroke during a short-term follow-up of 3 months. This case report was prepared in accordance with the CARE (CAse REport) guidelines, and the completed CARE checklist is provided as online supplementary material (for all online suppl. Material, see https://doi.org/10.1159/000553014).

Case Presentation

This is the case of a 55-year-old right-handed male with a medical history of hypertension. He had no prior history of stroke or similar neurological presentations. His home medications included ezetimibe 10 mg once daily, amlodipine 5 mg twice daily, and aspirin 81 mg once daily. He presented to the emergency department with sudden-onset dysarthria accompanied by left-sided weakness and hypoesthesia, for which the stroke code was activated.

A non-contrast brain computed tomography (CT) demonstrated chronic microangiopathic changes. There was no evidence of acute large territory infarction, intracranial hemorrhage, space-occupying lesion, mass effect, or midline shift. CT angiography (CTA) revealed atherosclerotic disease involving the left distal common carotid artery/internal carotid artery (ICA) origin, resulting in severe stenosis. The left ICA was occluded/thrombosed along its entire course, with a patent left middle cerebral artery supplied via the anterior communicating artery. The right ICA and right middle cerebral artery were patent, with no evidence of calcification, mural thickening, or other features of atherosclerotic disease, and all other major intracranial arteries were patent (shown in Fig. 1a).

On assessment, the patient's National Institutes of Health Stroke Scale (NIHSS) score was 4, comprising 1 point for left minor facial palsy, 2 points for left arm weakness with the limb unable to maintain position against gravity (drifting down to the bed with some residual effort), and 1 point for mild dysarthria. There was no aphasia, neglect, gaze palsy, or visual field deficit; the language deficit was pure dysarthria. Mechanical thrombectomy was not pursued as the right ICA was patent on CTA, and the left ICA occlusion was considered likely asymptomatic, as dysarthria is a non-localizing sign. Intravenous thrombolysis was also withheld as the patient was demonstrating rapid clinical improvement. The plan was for admission to the stroke unit for close neurological monitoring and initiation of dual antiplatelet therapy, with activation of the stroke code and repeat CT and CTA imaging in the event of neurological deterioration. Other laboratory investigations, electrocardiography, and transthoracic echocardiography were unremarkable.

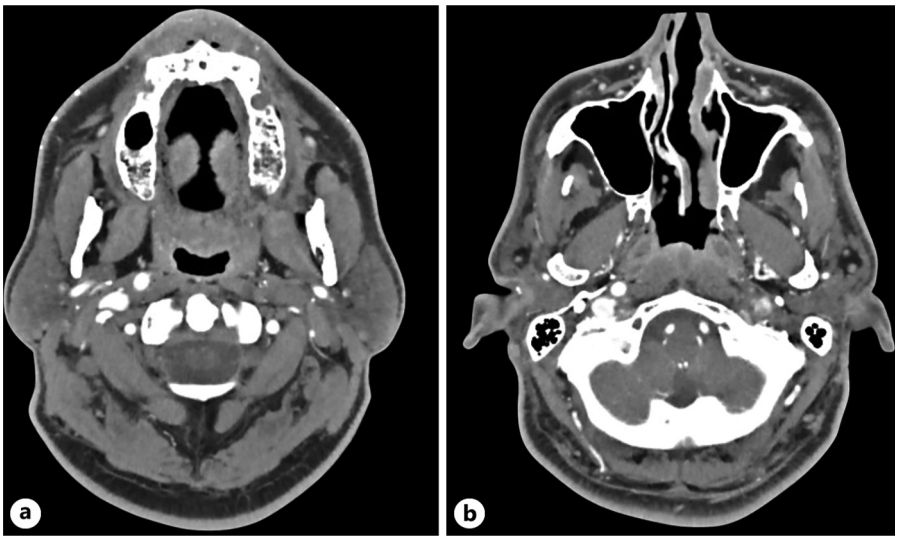


Fig. 1. Axial CT angiography of the neck. **a** Initial CTA on presentation. **b** Repeat CTA on day 5 of admission, at the time of clinical deterioration, showing no interval change.

Over the following days of hospitalization, the patient's neurological status improved, with the NIHSS score decreasing to 2 by the second day of admission (residual mild left facial asymmetry, 1 point, and persistent mild dysarthria, 1 point; motor power had returned to 5/5 in all extremities, sensation was intact, and there were no other focal deficits). MRI of the brain, including DWI (shown in Fig. 2a), demonstrated no acute infarction; FLAIR sequences showed scattered periventricular and subcortical white-matter hyperintensities consistent with chronic microangiopathic changes. Gray-white matter differentiation was preserved. There was no evidence of acute ischemia or acute intracranial hemorrhage. Throughout days 2–4 of admission, the patient remained neurologically stable at NIHSS 2, with normal motor power, sensation, gaze, and level of consciousness on serial focused examinations. Continued admission during this period reflected the heightened need for close in-hospital monitoring given the patient's limited collateral reserve from the chronically occluded contralateral left ICA, alongside the time required to complete extended cardiac rhythm monitoring and finalize the etiologic evaluation before a definitive treatment plan could be made.

On the fifth day of admission, a focused neurological examination earlier that day confirmed unchanged findings (NIHSS 2); the patient later that day developed sudden-onset left-sided weakness and worsening dysarthria. NIHSS score rose to 8, comprising 1 point for left facial palsy, 3 points for left arm weakness (no effort against gravity), 1 point for left leg motor weakness, 1 point for new left hemibody mild sensory loss, and 2 points for severe dysarthria; there was no new aphasia, neglect, gaze palsy, or visual field deficit. Repeat non-contrast brain CT (shown in Fig. 3) and CTA (shown in Fig. 1b) demonstrated no interval change from the previous imaging studies. Intravenous thrombolysis was not administered due to the rapid clinical improvement observed thereafter. A follow-up brain MRI performed the next day similarly showed no change compared with the prior examination (shown in Fig. 2b).

As part of the ongoing evaluation for the cause of recurrent TIAs, the case was discussed in a multidisciplinary team meeting, prompted by recurrence of ischemic symptoms within 1 week despite optimal medical therapy (indicating treatment failure and a high ongoing stroke risk) and the patient's vulnerability to additional ischemic or

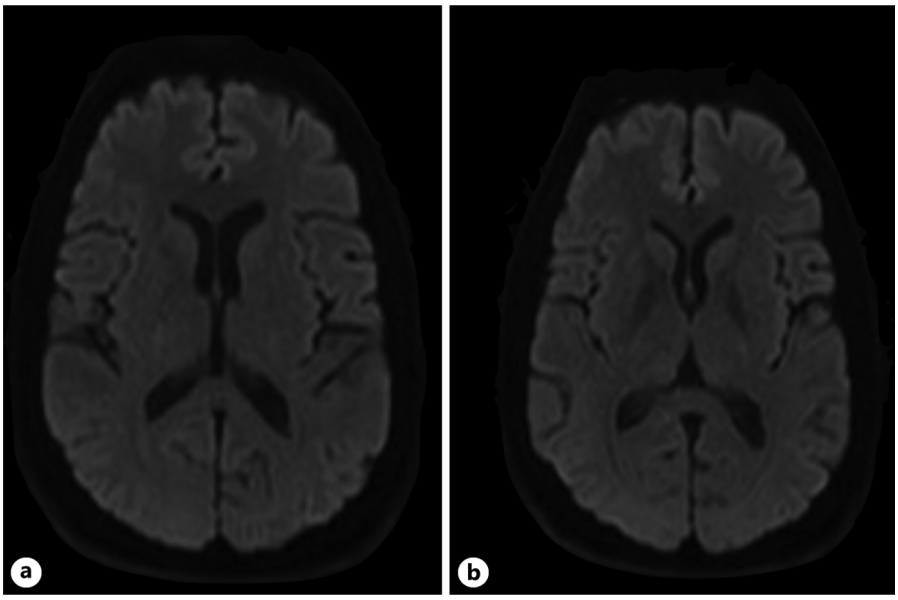


Fig. 2. Axial diffusion-weighted brain MRI. **a** Initial MRI on admission. **b** Follow-up MRI obtained the day after the recurrent ischemic event, demonstrating no acute diffusion restriction in either study.

hypoperfusion injury given the chronically occluded contralateral left ICA. Digital subtraction angiography (DSA) was elected to combine maximal diagnostic sensitivity for a subtle culprit lesion not visible on prior CTA with the option of immediate same-session endovascular treatment if one were identified. DSA revealed a focal shelf-like intraluminal projection arising from the posterior wall at the origin of the right internal carotid artery, consistent with a carotid web, causing greater than 50% luminal stenosis (shown in Fig. 4). Dynamic angiographic images demonstrated contrast pooling distal to the lesion, a finding considered characteristic of carotid web. In the same session, and via a right common femoral artery approach, carotid angioplasty and stenting were successfully performed without immediate complications. The patient was commenced on clopidogrel for 3 months in addition to lifelong aspirin therapy.

A carotid arteries duplex ultrasonography (US) performed for follow-up, it demonstrated the recently placed stent in the right ICA with residual moderate stenosis (shown in Fig. 5). At the 1-month and 3-month follow-ups, the patient reported no recurrence of symptoms and showed significant clinical improvement.

Discussion

A carotid web, first described in 1968 by Rainer et al. [4] is an intimal shelf in the carotid bulb that has been associated with ischemic events in young patients. Histologically, a carotid web represents a focal variant of intimal fibromuscular dysplasia, characterized by a thin fibrous projection extending from the posterior wall of the carotid bulb into the arterial lumen. The shelf-like protrusion disrupts blood flow, creating areas of stasis that predispose to thrombus formation [4, 5].

Importantly, carotid web-related stroke is mechanistically distinct from symptomatic atherosclerotic carotid stenosis. Stroke risk in carotid web is driven primarily by stasis and thrombus formation in the pouch immediately distal to the shelf, rather than by



Fig. 3. Axial CT of the head showing no acute major infarction or hemorrhage.

hemodynamically significant luminal narrowing; consequently, the degree of luminal stenosis does not reliably predict embolic risk in this entity [1, 6, 7]. Although most reported carotid webs produce only mild luminal narrowing, a systematic review of patients with carotid web from 37 publications showed that 16% of patients had ≥50% carotid stenosis [8].

Carotid web can be evaluated using US, CTA, MRI, and DSA. Although DSA remains the gold standard for its superior spatial and temporal resolution [9, 10], several studies report CTA to be similarly reliable [6, 10]. US is commonly used as the first-line modality for extracranial carotid assessment because it is rapid, widely available, and radiation-free. It provides both morphological and hemodynamic information and may even outperform CTA in demonstrating structural changes and hemodynamic evidence of thrombus, though its operator dependence can lead to misclassification as atherosclerosis [11, 12].

CTA offers rapid, high-resolution, multiplanar imaging that can identify carotid web and associated thrombus while excluding atherosclerosis or vascular injury. Its limitations include radiation, iodinated contrast exposure, and lack of flow assessment. On CTA, a carotid web typically appears as a membrane-like projection on axial views and a shelf-like filling defect on sagittal views [6, 13]. High-resolution MRI similarly provides detailed characterization of web morphology, often demonstrating a false lumen bifurcation and slow flow or pooling distal to the lesion, reflecting altered hemodynamics [14].

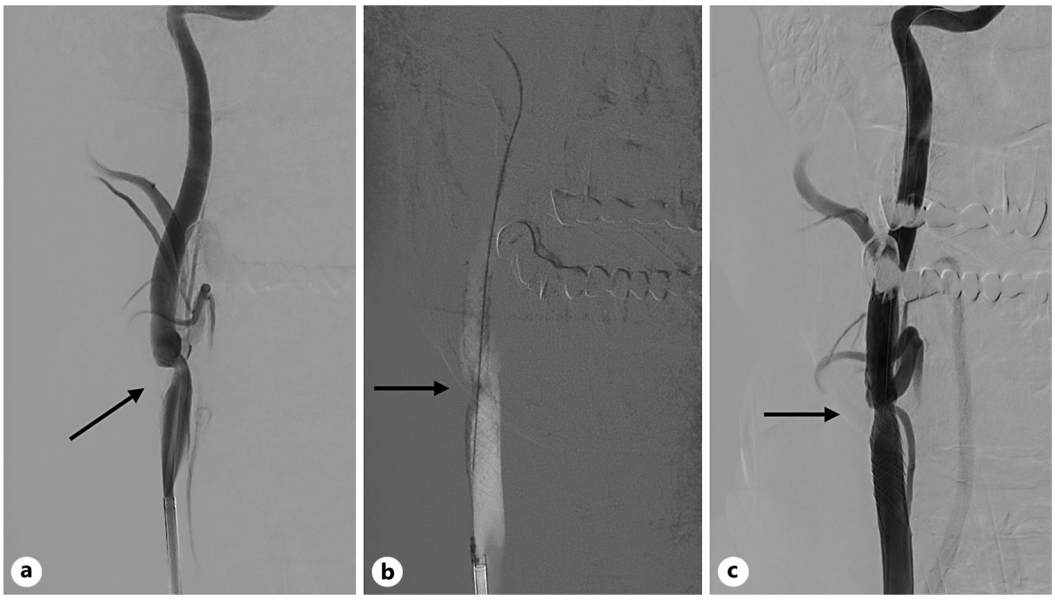


Fig. 4. Digital subtraction angiography (DSA) images illustrating the carotid web and stent placement. **a** Carotid web at the origin of the internal carotid artery. **b** Stent deployment across the lesion. **c** Post-deployment contrast flow demonstrating improved luminal patency.

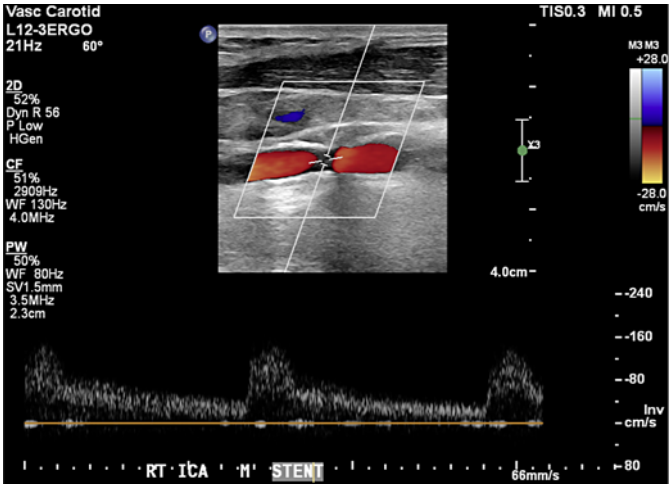


Fig. 5. Carotid duplex US showing the newly placed stent.

DSA typically shows a linear intraluminal filling defect with distal contrast pooling. Despite excellent resolution, its invasiveness and radiation/contrast requirements are notable drawbacks. In our case, the carotid web could only be identified on DSA. With the increasing availability of duplex US, CTA, and MRI, a multimodality approach has been shown to enhance diagnostic accuracy by providing complementary strengths and a reliable strategy for diagnosing carotid webs [10, 13, 14].

Distinguishing carotid web from focal atherosclerotic plaque with a shelf-like configuration is a recognized diagnostic challenge that merits explicit consideration in this case. The two entities are mechanistically distinct: carotid web represents focal intimal fibromuscular dysplasia and causes stroke through embolic mechanisms (stasis and

thrombus formation in the post-web pouch), whereas symptomatic atherosclerotic stenosis causes stroke through plaque rupture, atheroembolism, and, when severe, hemodynamic compromise [1, 2]. Imaging features favoring carotid web include a thin, focal, shelf-like contour arising perpendicular to the posterior wall of the carotid bulb; the absence of eccentric mural thickening or intramural calcification; and contrast pooling distal to the lesion on dynamic angiography [6, 10, 14]. The two entities can also coexist, with histopathologically confirmed cases reported [15].

In the present case, the lesion was identified on DSA at the origin of the right ICA, with these features present and no atherosclerotic findings in the right carotid bulb on CTA, in contrast to the contralateral left carotid bulb, which demonstrated extensive atherosclerotic disease with ICA occlusion. Together with the recurrent embolic clinical pattern on dual antiplatelet therapy and the absence of an alternative embolic source on echocardiography and rhythm assessment, these findings support carotid web as the symptomatic culprit lesion. Definitive distinction nevertheless requires histopathologic confirmation, which is unavailable when revascularization is performed by stenting rather than endarterectomy.

The treatment modalities for carotid webs typically involve medical therapy with or without revascularization. Medical monotherapy is often inadequate due to high recurrence risk. Joux et al. [16] reported a 30% stroke recurrence with antiplatelets, Haussen et al. found 32% recurrence [7], and Zhang et al. noted a 56% recurrence rate with a median time of 12 months [8].

Revascularization with carotid endarterectomy (CEA) or CAS is the primary treatment for carotid web and more effective in preventing stroke recurrence. In a systematic review of 37 studies encompassing 158 patients, Zhang et al. [8] reported no recurrent strokes or procedure-related complications among the 70 patients treated with CEA or CAS (50% each). While both appear efficacious, the choice between CEA and CAS remains debated. CEA is more invasive and may be unsuitable for high-risk patients, whereas CAS offers lower surgical risk, shorter hospitalization, and faster recovery. The 2023 European Society for Vascular Surgery (ESVS) guidelines suggest that in symptomatic patients with carotid web and no other stroke cause, CEA or CAS may be considered to prevent recurrence [1, 11]. This recommendation is based on limited Level C evidence, highlighting the need for further research. Currently, management remains individualized, considering patient preferences, anatomical factors, and associated comorbidities. In the presented case, medical monotherapy proved insufficient, with symptom recurrence occurring within 1 week. CAS was performed immediately after carotid web identification on DSA, enabling prompt treatment without additional anesthesia.

This case report offers key insights, highlighting the diagnostic challenge posed by left ICA chronic occlusion, which complicated identifying the culprit lesion. Immediate stenting during the same DSA session demonstrates a practical, efficient management approach that may reduce the risk of further ischemic events compared to elective treatment.

Limitations

This report has several limitations. The diagnosis was based on DSA imaging without histopathologic confirmation, as revascularization was performed by stenting rather than endarterectomy; atherosclerotic plaque with shelf-like configuration therefore cannot be entirely excluded. The patient also had contralateral atherosclerotic disease and vascular risk factors, although the chronically occluded left ICA and right-sided clinical-radiological pattern support the right-sided lesion as the symptomatic culprit. Finally, follow-up was limited to 3 months, and longer term surveillance is needed to confirm durability and detect late in-stent restenosis.

Conclusion

Carotid web is an underrecognized cause of recurrent ischemic events. Diagnosis requires high clinical suspicion and multimodality imaging, with DSA remaining the gold standard, although careful attention to morphologic features is essential to distinguish carotid web from atherosclerotic plaque with shelf-like configuration. Medical therapy alone carries a high risk of recurrence, whereas revascularization with CAS or CEA appears to provide effective secondary prevention with a favorable short-term outcome, highlighting revascularization as a key therapeutic option in symptomatic patients with carotid web. Larger studies and longer term follow-up are needed to refine noninvasive diagnostic criteria, and to define optimal long-term management.

Statement of Ethics

Madinah General Hospital at King Salman Medical City waived the need for IRB approval due to the absence of patient identification in the study's enrolled participant. Written informed consent was obtained from the patient for publication of the details of their medical case and any accompanying images. A copy of the signed consent form is available for review by the journal.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

M.A., T.K., I.I., and S.A. contributed equally to this work. All authors made substantial contributions to the conception, drafting, and revision of the manuscript. All authors read and approved the final manuscript.

Data Availability Statement

Relevant data generated or analyzed during this study is included in the article. For further inquiries, contact the corresponding author.

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