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Case Report

Carotid Web As A Rare Cause of Recurrent Ischemic Stroke: A Hemodynamic Phenomenon without Thromboembolism

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Abstract

This case report describes an unusual presentation of a carotid web (CaW) causing hemodynamic stroke in a 63-year-old hypertensive male, diverging from the typical thromboembolic mechanism commonly seen in younger patients. The patient presented with recurrent transient neurological deficits and was found to have left-sided watershed infarcts. Advanced imaging confirmed a CaW at the left carotid bulb causing severe flow restriction, evidenced by turbulent flow ("whirlpool sign") on Doppler and dynamic obstruction on Digital Subtraction Angiography (DSA), notably without thrombus formation. The patient was successfully treated with endovascular stenting of the left internal carotid artery (ICA), which restored normal cerebral blood flow and resulted in complete resolution of symptoms at the 3month follow-up. This case highlights the clinical variability of carotid webs, emphasizing that they can cause recurrent strokes via hemodynamic compromise rather than embolism, and confirms stenting as an effective management strategy for this atypical presentation.

Keywords: Ischemic Stroke; Carotid Web (CaW); Digital Subtraction Angiography (DSA)

Introduction

Ischemic stroke is a significant cause of morbidity and mortality worldwide; a substantial proportion of cases lack a clear etiology and are classified as cryptogenic stroke [1]. A crucial, though often under-recognized, cause of cryptogenic and recurrent ischemic stroke— particularly in younger, otherwise healthy patients—is the carotid web (CaW) [2]. The carotid web is defined as a thin layer of proliferative intimal tissue, typically 1–2 mm thick, that

originates from the arterial wall and projects into the vessel lumen. It is characteristically found at the carotid bulb or the proximal internal carotid artery (ICA) [3–5].

Since its initial description in 1968, the carotid web has been recognized as a high-risk factor for stroke, with a reported incidence of approximately 1.2% among ischemic stroke patients [5]. Its low incidence and potential for misdiagnosis—often confused with dissection flaps or atheromatous plaques—mean it frequently goes undetected, predisposing affected patients to recurrent events [6].

The present case is distinctive in that the carotid web led to significant hemodynamic obstruction rather than the typical thromboembolic pathway observed in most reported cases. It underscores the clinical and mechanistic variability of this condition and highlights the critical role of advanced imaging—including DSA, CTA, and Doppler ultrasound—in accurate diagnosis and treatment planning. The patient's excellent outcome after endovascular stenting further demonstrates the efficacy of this intervention for hemodynamically significant carotid web disease.

Case Presentation

A 63-year-old, right-handed male with a history of chronic hypertension presented to the outpatient department with severe giddiness and transient dysarthria. Two months prior, he had experienced right-sided transient hemiparesis and was managed externally as a left frontoparietal infarct. At that time, dual antiplatelet therapy and statins were initiated, and an outside CT brain angiography was reported as unremarkable.

On current presentation, blood pressure was significantly elevated at 190/90 mmHg. Neurological examination was otherwise unremarkable (NIHSS score of 0), consistent with a transient ischemic attack (TIA) or a minor recurrent stroke.

Imaging and diagnostic findings

Initial CT and MRI of the brain revealed chronic watershed infarcts in the left ACA–MCA and left.

MCA–PCA territories (Figure 1), indicating ischemic injury driven by cerebral hypoperfusion rather than large-vessel occlusion. Advanced vascular studies were undertaken to identify the underlying cause:

- **CT Angiography (CTA):** Axial and sagittal CTA images of the neck demonstrated a thin, shelf-like linear hypodensity proximal to the left carotid bifurcation within the carotid bulb, highly consistent with a carotid web (Figure 2A, 2B).
- **Doppler Ultrasound**: Confirmed the presence of the carotid web at the left ICA origin. Axial (Figure 3A) and longitudinal (Figure 3B) planes demonstrated an echogenic linear stripe across the lumen without associated calcification or thrombus. Color Doppler (Figure 3C) revealed the hallmark "whirlpool sign," indicating severe turbulent flow at the site of the web.
- **Digital Subtraction Angiography (DSA):** Confirmed the carotid web in the distal common carotid artery (CCA) (Figure 4A), with dynamic obstruction to flow in both the ipsilateral ICA and external carotid artery (ECA) (Figure 4B). Crucially, no thrombus was identified. Pre-stenting intracranial angiography demonstrated compensatory cross-filling of the left intracranial circulation from the contralateral ICA (Figure 6A), confirming that the patient's watershed infarcts were the result of hemodynamic failure rather than embolism (Figures 6–7).

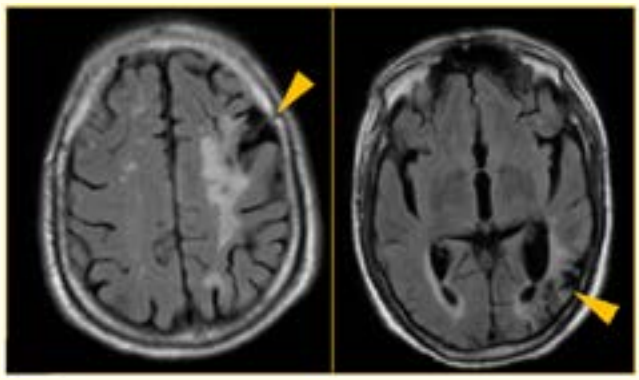

Figure 1: Axial FLAIR MRI images demonstrating (A) gliotic changes in the left frontoparietal region consistent with prior ACA–MCA watershed infarction, and (B) gliotic changes in the left temporo-occipital region consistent with MCA–PCA watershed infarction. These findings indicate chronic hemodynamic ischemic injury in the arterial border zones.

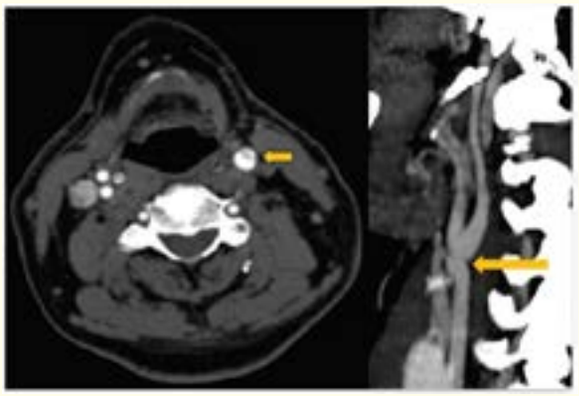

Figure 2: Computed tomography angiography (CTA) of the neck. (A) Axial and (B) sagittal reformatted images demonstrate a thin, shelf-like hypodensity (arrows) proximal to the left carotid bifurcation within the carotid bulb, consistent with a carotid web. Note the absence of calcification or wall thickening.

Figure 3: Doppler ultrasound of the left carotid bulb. (A) Axial and (B) longitudinal grayscale images demonstrate an echogenic linear stripe (arrows) across the lumen at the origin of the left ICA, without associated calcification or thrombus. (C) Color Doppler imaging reveals a characteristic swirling "whirlpool" flow pattern, indicating turbulence at the site of the web—a hemodynamic hallmark of significant carotid web disease.

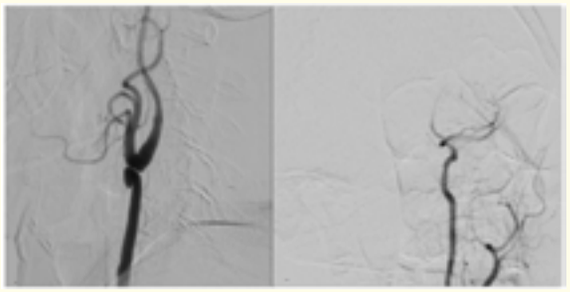

Figure 4: Digital subtraction angiography (DSA). (A) Oblique projection demonstrating the carotid web as a shelf-like filling defect in the distal CCA. (B) Anteroposterior projection showing dynamic obstruction to flow in the ipsilateral ICA and ECA. No thrombus is visible within or distal to the web.

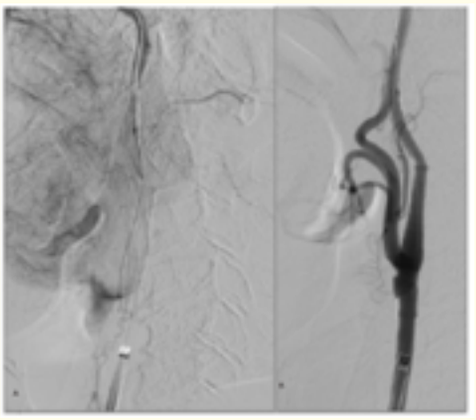

Figure 5: DSA images documenting the endovascular procedure. (A) Guiding sheath positioned proximal to the carotid web prior to stent deployment. (B) A 6–8 mm × 40 mm tapered stent deployed across the web from the left ICA into the CCA after crossing with a 0.018-inch microwire, with restored luminal patency.

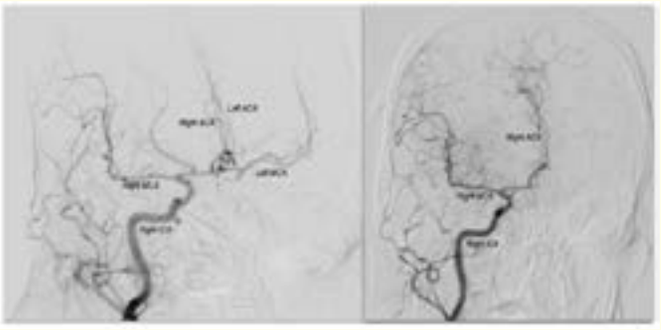

Figure 6: Intracranial DSA. (A) Pre-stenting right common carotid artery injection demonstrating cross-filling of the left intracranial circulation (left MCA and ACA) via the contralateral ICA, confirming critically reduced left ICA flow. (B) Post-stenting injection showing restoration of normal antegrade left intracranial flow.

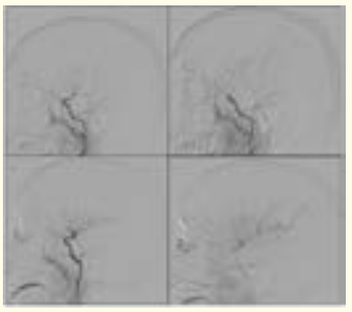

Figure 7: Comparison of left CCA intracranial angiograms before stenting (A, B) and after stenting (C, D). Post-stenting images demonstrate markedly improved antegrade flow in the left ICA and its intracranial branches, with elimination of the compensatory contralateral cross-flow pattern.

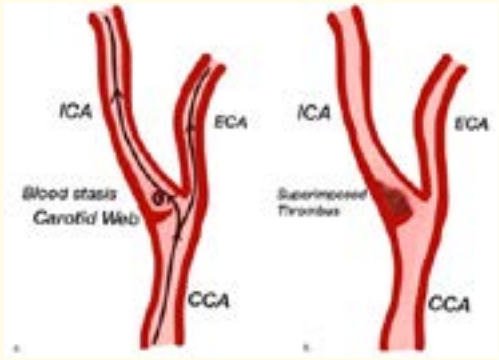

Figure 8: Schematic illustration of carotid web pathophysiology. (A) Luminal protrusion of proliferating myofibers disrupts laminar flow, creating a zone of blood stasis. (B) Persistent flow disturbance promotes mural thrombus formation at the site of the web, which may subsequently embolize to cause ipsilateral ischemic stroke.

Collectively, these findings established a diagnosis of hemodynamic stroke secondary to a flow restricting carotid web, without evidence of thromboembolism.

Intervention and outcome

Given the documented dynamic obstruction and recurrent neurological symptoms, endovascular intervention was performed. A guiding sheath was placed proximal to the carotid web, the web was crossed with a 0.018-inch microwire, and a tapered stent (6–8 mm × 40 mm) was deployed from the left ICA across the web into the CCA (Figure 5A, 5B). Post-stenting angiography confirmed complete normalization of blood flow in the left ICA with no residual obstruction (Figures 6B, 7C, 7D). The patient was discharged without neurological deficit and remained entirely symptom-free at the 3-month follow-up.

Discussion

Pathophysiology and mechanism of stroke

Pathologically, the carotid web is considered a specific variant of fibromuscular dysplasia (FMD) involving intimal hyperplasia. Longitudinal sectioning reveals vascular intimal proliferation accompanied by fibrosis and myxoid degeneration, forming a valve-like structure that protrudes into the vessel lumen—in contrast to classic FMD, which primarily affects the medial layer [7]. The exact pathogenesis remains unclear but is thought to involve a combination of congenital predisposition and acquired factors, including vascular injury and hormonal influences such as those associated with oral contraceptive use [8]. The aberrant tissue protrusion disrupts laminar flow, generating a zone of stasis and turbulence distal to the defect—a thrombogenic niche well characterized by computational fluid dynamics (CFD) studies demonstrating an extensive recirculation zone at the distal margin of the web [9]. This flow disturbance promotes mural thrombus formation; subsequent thrombus fragmentation and embolization typically affect the ipsilateral MCA or ACA territories [10]. Available data indicate that 12–29% of stroke patients with a carotid web demonstrate overlapping thrombosis [3].

The present case diverges from this typical thromboembolic paradigm. The carotid web was sufficiently occlusive to produce dynamic obstruction visible on DSA and confirmed by the whirlpool sign on Doppler, without any identifiable thrombus. The resultant watershed infarcts— ischemic injury in the hemodynamically vulnerable border zones between major arterial territories—are the clinical hallmark of hypoperfusion, not embolic large-vessel occlusion.

Table 1 summarizes the key differences between this presentation and the typical carotid web patient profile.

Feature	Typical Carotid Web Patients	Present Case
Thrombus Formation	Common; leads to embolism and infarcts	Absent; no thrombus identified on any modality
Sex	Predominantly female	Male
Age	Younger (mean age ~38.3 years)	Older (63 years)
Vascular Risk Factors	Rare (low prevalence of hypertension, diabetes, etc.)	Hypertension (BP 190/90 mmHg)
Stroke Type	Embolic strokes in MCA/ACA territories	Watershed infarcts (hemodynamic mechanism)

Recurrence Rate	High short-term recurrence (~83%)	No recurrence at 3-month follow-up
Infarct Pattern	Typically single large vessel territory	Bilateral watershed distribution
Diagnostic Findings	Thrombus or emboli on imaging	Dynamic obstruction only; no thrombus
Treatment Focus	Thromboembolic prevention (antiplatelets/ anticoagulants)	Hemodynamic restoration via endovascular stenting

Table 1: Typical Imaging and Clinical Features of Carotid Web: Comparison with the Present Case.

Imaging features and differential diagnosis

Accurate diagnosis requires advanced multimodal imaging, as the subtle morphology of the web renders it susceptible to misdiagnosis or omission on routine studies.

Imaging modalities

CT Angiography (CTA) is currently the recommended first-line diagnostic modality, offering detection rates comparable to DSA while being relatively safe, widely available, and non-invasive. On CTA multiplanar reformats, the web appears as a triangular, linear, or membranous filling defect projecting from the posterior wall of the carotid bulb or proximal ICA. Oblique sagittal views are considered optimal for identification given the web's film-like thinness (1–2 mm). CTA also facilitates differentiation from other pathologies such as carotid dissection or aneurysm and can identify associated thrombosis based on density differences [12].

Digital Subtraction Angiography (DSA) remains the gold standard for dynamic assessment of flow. The carotid web typically appears as a linear or shelf-like filling defect with less luminal stenosis than that seen in atherosclerosis; contrast pooling distal to the web may be observed during the late venous phase. Because the web is frequently located on the posterior wall, twoprojection imaging alone (anteroposterior and lateral) may result in misdiagnosis; additional oblique projections should be obtained when clinical suspicion is high [13].

Magnetic Resonance Imaging (MRI) and MR angiography (MRA) depict the web as a film-like structure from the posterior carotid wall, with morphology generally consistent with CTA findings. Phase-contrast and time-of-flight MRA are radiation-free alternatives useful in pregnant patients or those with renal insufficiency, though MRA generally has lower sensitivity and specificity compared to CTA and DSA. Vessel wall MRI (VW-MRI) provides multiplanar structural characterization; on VW-MRI, the web may appear isointense on T1-weighted sequences and slightly hyperintense on fat-suppressed T2-weighted sequences, with significant post-contrast enhancement [13].

Doppler Ultrasound is less expensive and more widely available but carries a higher risk of misdiagnosis, including confusion with carotid dissection or ulcerative plaque. The web appears as an echogenic intimal projection from the posterior carotid bulb. Importantly, Doppler ultrasound is valuable for hemodynamic assessment, often demonstrating the characteristic "whirlpool sign" (turbulent eddy flow) at the angle between the web and vessel wall. Contrastenhanced and three-dimensional ultrasound techniques may reduce the misdiagnosis rate [13].

Differential diagnosis

A critical diagnostic distinction must be drawn between the carotid web and other vascular pathologies. Table 2 summarizes the key imaging features that differentiate the carotid web from its most common mimics.

Pathology	Distinctive Imaging Features
Carotid Web	Thin, non-calcified, non-atheromatous filling defect (1–2 mm), projecting from the posterior wall of the carotid bulb. No focal calcification or wall thickening.
Focal Atherosclerotic Plaque	Irregular, often calcified or hypoattenuating wall thickening causing variable luminal stenosis; may be circumferential.
Carotid Dissection	Irregular borders, distal propagation from the bulb, intramural hematoma, or classic "double lumen" appearance.
Fibromuscular Dysplasia (FMD)	Multisegmental involvement producing the characteristic "string of beads" pattern affecting primarily the media [16].

Table 2: Differential Diagnosis of Carotid Web and Common Mimics

In patients with confirmed atherosclerotic plaque, intraplaque hemorrhage (IPH) volume is the single most important predictor of plaque instability and recurrent infarction; patients with recurrent cerebral infarction demonstrate significantly higher IPH volume and percentage compared to those with an initial event [12].

Management strategies

No consensus currently exists regarding the optimal management of carotid web. Available approaches include:

Pharmacological therapy: Dual antiplatelet or anticoagulant regimens are used in the acute setting. However, single antiplatelet therapy has proven insufficient for secondary prevention given the predominantly flow-mediated embolic mechanism. Reported recurrence rates with conservative pharmacological management approach 30% [17].

Endovascular and surgical intervention: For symptomatic carotid webs, carotid endarterectomy or carotid artery stenting is favored. These procedures eliminate the hemodynamic disturbance and have been associated with favorable outcomes and low recurrence rates in reported series [17-19].

In the present case, endovascular stenting was selected given the documented dynamic obstruction and recurrent symptoms despite antiplatelet therapy. The complete normalization of flow and absence of recurrence at 3-month follow-up support stenting as an effective treatment for hemodynamically significant carotid web disease. Larger prospective studies are needed to standardize management guidelines.

Conclusion

The carotid web is an important and under-recognized cause of cryptogenic and recurrent ischemic stroke that warrants active consideration, particularly in patients presenting with unexplained or recurrent events. Early recognition is critical: the characteristic imaging appearance—a thin, non-calcified shelf-like filling defect at the carotid bulb—can be readily identified on CTA and DSA when specifically sought, yet is frequently overlooked or misattributed to atherosclerosis or dissection.

The present case contributes meaningfully to the literature by demonstrating that the carotid web can cause severe hemodynamic obstruction and watershed infarction without any thromboembolic component—a mechanism that is distinctly uncommon and mandates a different therapeutic strategy. Management decisions must therefore be guided by the precise pathological mechanism, rather than by a uniform algorithmic approach. In this patient, endovascular stenting proved highly effective, restoring normal cerebral perfusion and preventing further neurological events.

Looking ahead, systematic screening for carotid web should be considered in patients with recurrent unexplained ischemic stroke, and future research should prioritize prospective studies to standardize management guidelines, define optimal indications for endovascular versus surgical intervention, and clarify the long-term durability of stenting across the full spectrum of carotid web presentations. Timely and accurate diagnosis, followed by an individualized treatment strategy, remains the cornerstone of effective management.

Ethics Approval and Consent to Participate

This study was conducted in accordance with the ethical standards of the institutional research committee and with the principles of the Declaration of Helsinki. As this is a single case report involving no experimental intervention beyond standard clinical care, formal ethics committee approval was not required per institutional policy. Written informed consent was obtained from the patient for participation and use of anonymized clinical and imaging data for academic and publication purposes.

Consent for Publication

Written informed consent for publication of clinical details and associated radiological images was obtained from the patient. A copy of the written consent is available for review by the Editor-in-Chief upon reasonable request.

Availability of Data and Materials

All relevant clinical data and imaging findings supporting the conclusions of this article are included within the manuscript. Additional anonymized data may be made available by the corresponding author upon reasonable request, in accordance with institutional and ethical guidelines.

Competing Interests

The authors declare no competing interests, financial or non-financial, related to this manuscript.

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Authors' Contributions

- Dr. Esha Meherwal: Data collection, imaging interpretation, literature review, manuscript drafting, and preparation of figures.
- Dr. Vivek Murumkar: Neurointerventional planning and procedure, critical revision of the manuscript, and supervision of radiological analysis.
- Dr. Priscilla Joshi: Conceptualization of the manuscript, academic supervision, and critical review of intellectual content.
- Dr. Sankar Prasad Gorthi: Clinical evaluation, neurological management, and review of the manuscript for clinical content.

All authors read and approved the final manuscript.

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