

Received: 2026.04.23
Accepted: 2026.09.03
Available online: 2026.10.06
Published: 2026.XX.XX

e-ISSN 1941-5923
© Am J Case Rep, 2026; 27: e953876
DOI: 10.12659/AJCR.953876

Carotid Web-Associated Ischemic Stroke in a Young Woman: Diagnostic Pitfalls and Endovascular Management

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

AEF 1 **Barbara Rosiak**
ABDE 1 **Adam Dobek** 
BCD 2,3 **Dagmara Wilmańska** 
ABCE 4 **Beata Śliwińska** 
ADG 4 **Jacek J. Rożniecki** 
ADG 5 **Mariusz Stasiotek** 
BCF 1 **Jarosław Arkadiusz Chrzastek** 
ADG 1 **Ludomir Stefańczyk**

1 I Department of Radiology and Diagnostic Imaging, Medical University of Łódź, Łódź, Poland
2 I Department of Radiology and Diagnostic Imaging, Norbert Barlicki Memorial Teaching Hospital No. 1, Medical University of Łódź, Łódź, Poland
3 III Department of Radiology and Diagnostic Imaging, Medical University of Łódź, Łódź, Poland
4 Department of Neurology, Stroke and Neurorehabilitation, Medical University of Łódź, Łódź, Poland
5 Department of Neurology, Medical University of Łódź, Łódź, Poland

Corresponding Author: Adam Dobek, I Department of Radiology and Diagnostic Imaging, Medical University of Łódź, ul. dr. Stefana Kopcińskiego 22/28, 90-153 Łódź, Poland, e-mail: adam.dobek@umed.lodz.pl
Financial support: None declared
Conflict of interest: None declared

Patient: Female, 40-year-old
Final Diagnosis: Carotid web-associated ischemic stroke
Symptoms: Right-sided weakness • speech disturbances
Clinical Procedure: —
Specialty: Neurology • Radiology

Objective: Unknown etiology
Background: Carotid web (CaW) is an uncommon intimal variant of fibromuscular dysplasia affecting the internal carotid artery (ICA) and is increasingly recognized as a cause of ischemic stroke in young and middle-aged patients without traditional vascular risk factors. The lesion is a thin, shelf-like fibrointimal projection arising from the carotid bulb that disrupts local hemodynamics and promotes thrombus formation and cerebral embolization. CaW is frequently underdiagnosed because of its subtle imaging appearance and limited clinical awareness.
Case Report: A 40-year-old woman presented with sudden speech disturbances and right-sided weakness. Neurological examination revealed mixed aphasia and mild right hemiparesis (National Institutes of Health Stroke Scale score of 8). Computed tomography angiography (CTA) demonstrated distal branch occlusion of the left middle cerebral artery and an abnormal lesion at the left carotid bulb, suspected to represent CaW. As the intravenous thrombolysis window had elapsed, mechanical thrombectomy was performed, achieving complete reperfusion (modified Thrombolysis in Cerebral Infarction grade 3). Follow-up imaging confirmed left frontoparietal ischemic changes. Extensive diagnostic evaluation excluded other causes of stroke. Persistent carotid stenosis was identified during follow-up, and the patient subsequently underwent carotid angioplasty with stent implantation, restoring normal arterial lumen and cerebral blood flow. The postoperative course was uneventful, and she was discharged in good clinical condition.

Conclusions: This case highlights the importance of considering CaW in the differential diagnosis of cryptogenic stroke in young patients. Advanced vascular imaging may facilitate timely diagnosis, while appropriate treatment may reduce recurrent cerebrovascular risk. Further studies are needed to define optimal diagnostic and therapeutic strategies for CaW.

Keywords: Carotid Artery Diseases • Stroke • Thrombectomy • Angioplasty
Full-text PDF: <https://www.amjcaserep.com/abstract/index/idArt/953876>

 2523  —  6  22



Publisher's note: All claims expressed in this article are solely those of the authors and do not necessarily represent those of their affiliated organizations, or those of the publisher, the editors and the reviewers. Any product that may be evaluated in this article, or claim that may be made by its manufacturer, is not guaranteed or endorsed by the publisher

Introduction

Carotid web (CaW) is an uncommon intimal variant of fibromuscular dysplasia affecting the internal carotid artery (ICA) and is increasingly recognized as a potential cause of ischemic stroke in young and middle-aged patients without conventional vascular risk factors [1-3]. The lesion is characterized by a thin shelf-like fibrointimal projection arising from the carotid bulb that disturbs local blood flow and promotes thrombus formation with subsequent artery-to-artery embolization [2,4].

Although its true prevalence remains uncertain, CaW is estimated to account for approximately 1% to 2% of all ischemic strokes and up to 10% to 20% of cryptogenic strokes in young adults, making its recognition clinically important despite its rarity [4,5]. Histopathological studies demonstrate fibroelastic intimal thickening without inflammatory infiltrates, lipid deposition, or disruption of the internal elastic lamina, distinguishing CaW from atherosclerotic plaque and arterial dissection [6-8].

Diagnosis may be challenging because the lesion is often subtle and can be misinterpreted as an ulcerated plaque or focal dissection on routine vascular imaging. Consequently, multi-modal vascular assessment is frequently required to establish the correct diagnosis and guide treatment decisions [2,6].

We present the case of a young woman with acute ischemic stroke in whom the diagnostic pathway evolved from an initial suspicion of ulcerated carotid plaque or focal dissection to the final diagnosis of CaW based on complementary vascular imaging. The case highlights the diagnostic pitfalls associated with this entity and illustrates staged management consisting of acute mechanical thrombectomy followed by delayed carotid artery stenting after exclusion of alternative stroke etiologies.

Case Report

In November 2025, a 40-year-old woman was admitted to the department of clinical neurology due to the sudden onset of speech disturbances. The symptoms developed in the morning hours on the day of admission and manifested as mixed aphasia and mild weakness of the right limbs. Prior to symptom onset, the patient had been fully independent in activities of daily living and professionally active (pre-stroke modified Rankin Scale score of 0). Her past medical history was notable for hypothyroidism treated with levothyroxine. She had also been diagnosed with attention-deficit/hyperactivity disorder, which was previously treated with methylphenidate and had been discontinued 6 months before hospitalization. The patient also used hydroxyzine as needed.

On admission, the patient was conscious and in stable general condition. Neurological examination revealed mixed aphasia with predominance of the motor component, mild right-sided facial nerve paresis, and mild weakness of the right upper and lower limbs (Lovett 4/5). No sensory deficits, coordination disturbances, or pathological reflexes were detected. The National Institutes of Health Stroke Scale (NIHSS) score was 8.

Non-contrast-enhanced computed tomography (CT) of the head revealed no acute abnormalities. CT angiography (CTA) demonstrated a short-segment contrast filling defect in a distal cortical branch at the M2/M3 junction of the left middle cerebral artery. Additionally, a non-calcified lesion approximately 10 mm in length with features suggestive of ulceration was identified within the posteromedial wall of the left ICA just above the carotid bulb, causing focal luminal narrowing (Figure 1A-1C). No other clinically relevant abnormalities were identified within the cervicocerebral circulation.

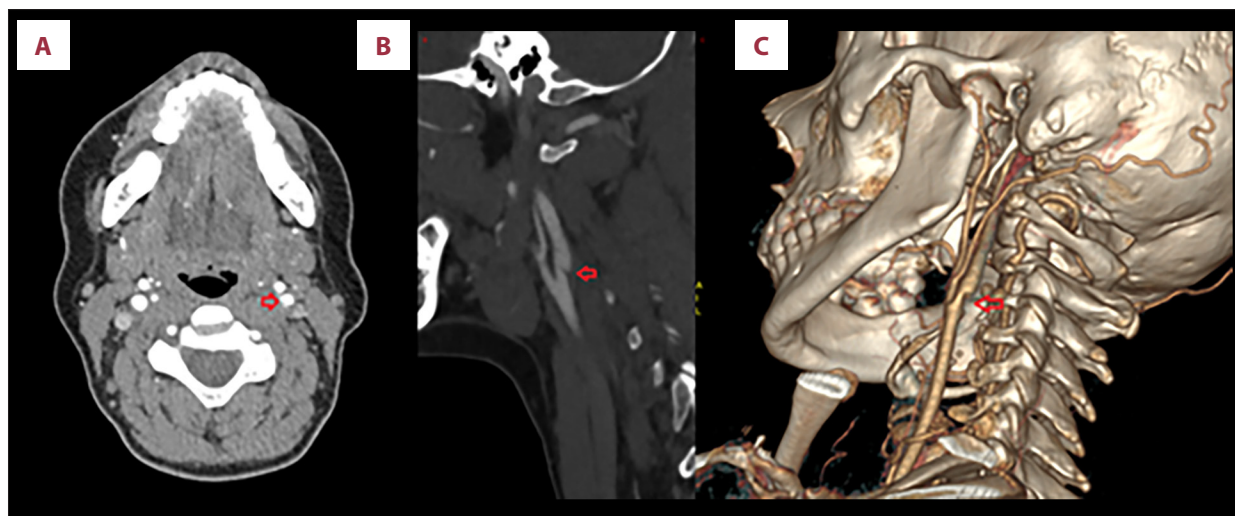


Figure 1. Lesion within the left internal carotid artery. (A) Axial computed tomography angiography (CTA) image. **(B)** Sagittal CTA image. **(C)** Three-dimensional CTA reconstruction. Arrows indicate the lesion.

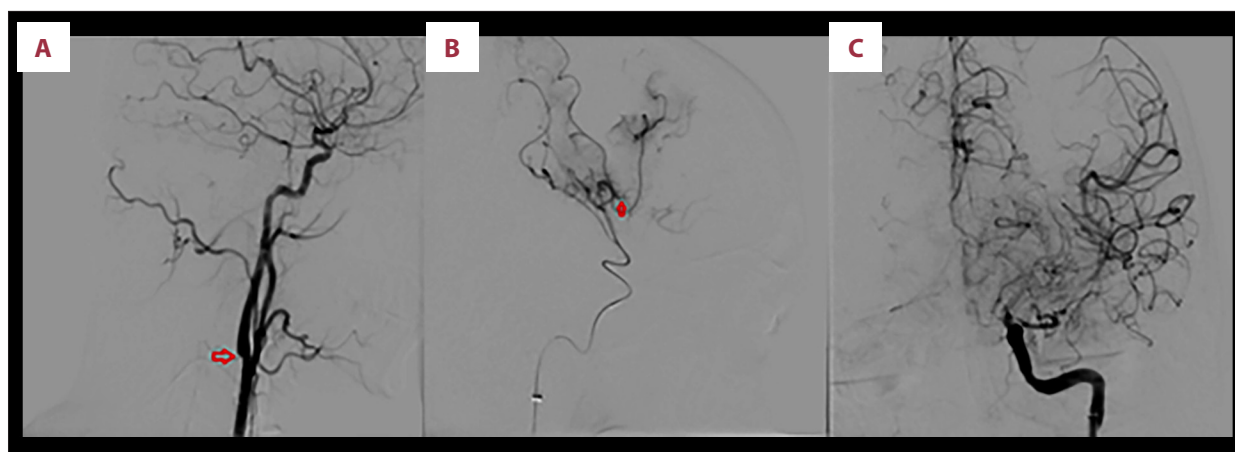


Figure 2. Angiographic images. (A) Focal luminal irregularity along the posteromedial wall of the proximal left internal carotid artery, immediately above the carotid bulb (arrow), initially interpreted during the emergency angiographic assessment as plaque ulceration. (B) Occlusion of an M3 branch of the left middle cerebral artery (arrow). (C) Status post recanalization of the M3 branch of the left middle cerebral artery.

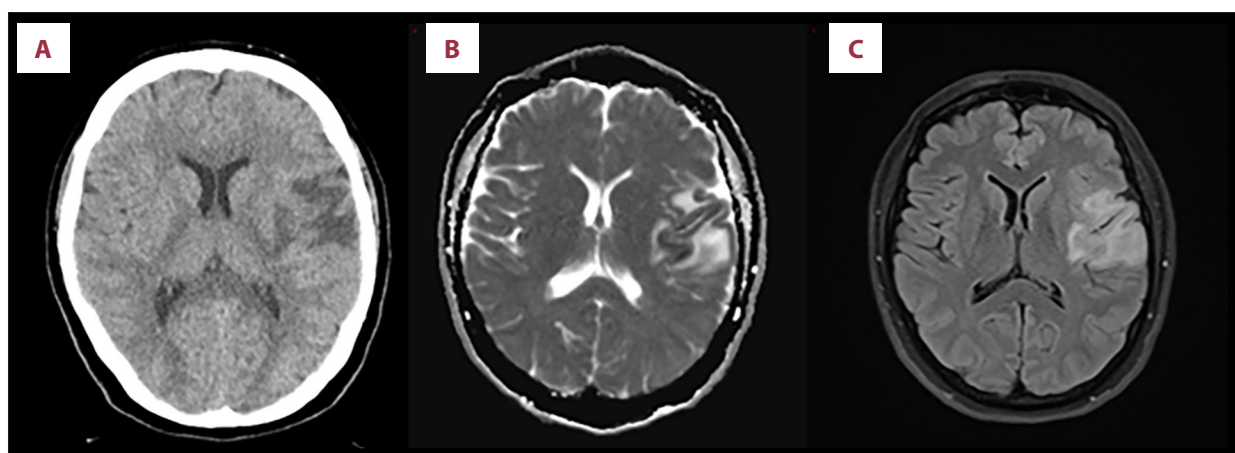


Figure 3. Follow-up neuroimaging demonstrating an evolving acute ischemic infarction. (A) Axial non-contrast-enhanced computed tomography (CT) obtained 24 hours after symptom onset shows an ill-defined area of cortical and subcortical hypoattenuation in the left frontoparietal region, with loss of normal gray-white matter differentiation and mild focal sulcal effacement, consistent with evolving acute ischemic changes. No hemorrhagic transformation or significant midline shift is evident on the presented image. (B) Axial apparent diffusion coefficient map demonstrates corresponding reduced apparent diffusion coefficient signal, indicating decreased water diffusivity secondary to cytotoxic edema and supporting the diagnosis of acute cerebral infarction. (C) Axial fluid-attenuated inversion recovery image shows concordant cortical-subcortical hyperintensity with mild local swelling, delineating the extent of the ischemic lesion more clearly than non-contrast-enhanced CT.

Because the therapeutic window for intravenous thrombolysis had elapsed, the patient underwent mechanical thrombectomy. Initial angiography confirmed occlusion of an M3 branch of the left middle cerebral artery and demonstrated the carotid bulb lesion, which was initially interpreted as an ulcerated plaque. The first aspiration pass resulted in partial recanalization; however, persistent occlusion of a larger M2 branch supplying a wider ischemic territory prompted a second pass, resulting in complete reperfusion (modified Thrombolysis in Cerebral Infarction [mTICI] grade 3) and restoration of flow within the left ICA territory (Figure 2A-2C). Immediately after

the procedure, weakness of the right lower limb resolved, while mild distal weakness of the right upper limb, central facial paresis, and motor aphasia partially improved. The NIHSS score decreased to 7.

Follow-up CT performed 24 hours after thrombectomy demonstrated acute ischemic changes involving the left frontoparietal region, including the insular cortex. Brain magnetic resonance imaging (MRI) performed 48 hours later was obtained to further characterize the extent of the ischemic injury and to exclude additional acute lesions. MRI confirmed the acute

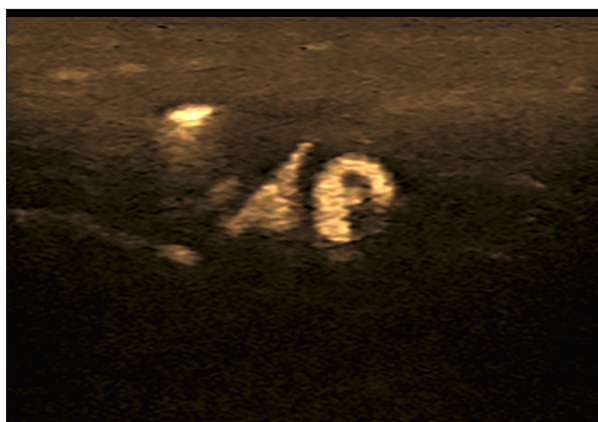


Figure 4. Axial B-flow ultrasound image demonstrating a shelf-like intraluminal projection in the left internal carotid artery, consistent with a carotid web.

infarction in the left frontoparietal region, including the insular cortex, without evidence of additional ischemic foci or other significant intracranial abnormalities (**Figure 3A-3C**).

To further characterize the carotid lesion, Doppler ultrasonography was performed and demonstrated a characteristic shelf-like intraluminal projection arising from the posterior wall of the carotid bulb, consistent with CaW (**Figure 4**).

The diagnostic interpretation of the carotid lesion evolved during hospitalization. Initial CTA and angiographic assessment suggested an ulcerated non-calcified plaque, while focal arterial dissection was also considered because of the patient's age and lesion morphology. However, Doppler ultrasonography demonstrated the characteristic shelf-like intraluminal projection typical of CaW. Furthermore, CTA showed no long-segment stenosis, double lumen, mural hematoma, or visible intimal flap, making arterial dissection unlikely. The absence of generalized atherosclerotic disease and conventional vascular risk factors further supported CaW as the most probable cause of stroke. To exclude alternative stroke etiologies, Holter ECG monitoring, transthoracic echocardiography, and transesophageal echocardiography were performed. No clinically significant arrhythmias, structural cardiac abnormalities, or patent foramen ovale were identified. Given the patient's young age, history of recurrent miscarriages, and heavy menstrual bleeding, the diagnostic workup was extended to include evaluation for autoimmune disorders and inherited or acquired thrombophilia. The laboratory assessment included antiphospholipid antibodies, lupus anticoagulant, ANA profile, ANCA antibodies, factor V Leiden mutation, protein C, protein S, factor II activity, and homocysteine level. All results were negative or within reference ranges, effectively excluding autoimmune disease and thrombophilia as potential causes of stroke.

At this stage, carotid artery stenting was intentionally deferred. Although CaW was considered the leading diagnosis, the patient

remained in the acute phase of ischemic stroke, and exclusion of alternative embolic sources was considered essential before definitive carotid intervention. This staged strategy allowed confirmation of the stroke mechanism and optimization of secondary prevention before elective revascularization.

During hospitalization, secondary stroke prevention with antiplatelet and statin therapy was initiated, together with motor rehabilitation and speech therapy. The patient experienced one transient worsening of focal neurological deficits accompanied by a focal motor seizure involving the right side, without evidence of a new vascular event. She was discharged home in good general condition with mild residual motor aphasia, slight right-sided weakness, and central facial paresis. Treatment with acetylsalicylic acid (75 mg daily), atorvastatin (40 mg daily), and levothyroxine was continued. Based on the imaging findings suggestive of CaW and the negative results of the extended diagnostic workup, the patient was qualified for elective carotid artery stenting. Dual antiplatelet therapy with acetylsalicylic acid and clopidogrel was initiated before the planned procedure.

In March 2026, the patient was readmitted for elective treatment of persistent left ICA stenosis. During the interval following the stroke, she reported only intermittent balance disturbances and presented without significant neurological deficits. Follow-up non-contrast-enhanced CT of the head demonstrated stable chronic post-ischemic changes without evidence of new ischemia or intracranial hemorrhage.

Angiography confirmed the previously identified lesion of the left ICA. Carotid artery stenting with implantation of a Roadsaver stent using distal neuroprotection was successfully performed. Immediate post procedural control angiography demonstrated full restoration of the vessel lumen and normal cerebral blood flow (**Figure 5A-5D**). To provide a clear overview of the management strategy, the complete diagnostic and therapeutic algorithm is illustrated in the flowchart (**Figure 6**).

Neurological examination after carotid artery stenting showed no focal neurological deficits, with preserved muscle strength and sensation, and no pathological findings. Her clinical status corresponded to a modified Rankin Scale score of 0. The post-operative course was uneventful, and follow-up ultrasonography of the puncture site revealed no vascular complications. She was discharged home with dual antiplatelet therapy consisting of clopidogrel 75 mg once daily for 90 days and acetylsalicylic acid 75 mg once daily, together with pantoprazole 40 mg once daily and rosuvastatin 10 mg once daily. Follow-up vascular imaging with Doppler ultrasonography was scheduled at 6 and 12 months after stenting to assess stent patency.



Figure 5. Sagittal angiographic images. (A) Before stent placement. (B) After stent implantation. (C) Angiographic visualization of blood flow through the stent and the internal carotid artery with its branches. (D) Blood flow in the distal branches of the internal carotid artery.

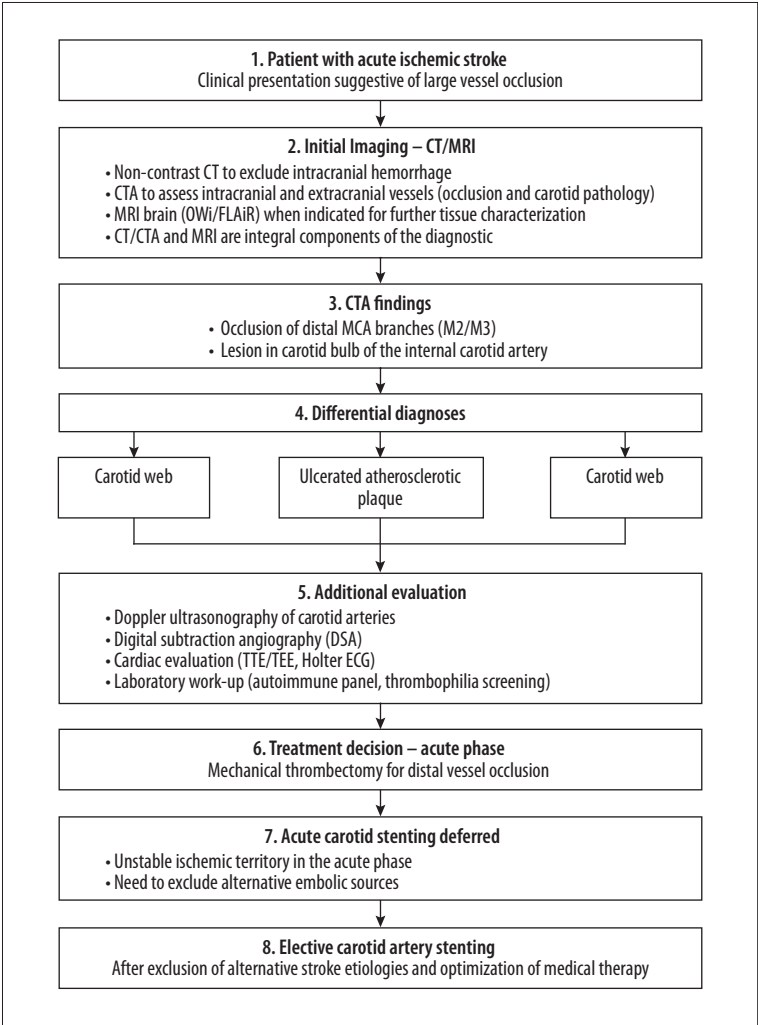


Figure 6. Stepwise diagnostic algorithm showing the evaluation of carotid web and the resulting treatment strategy.

Discussion

Ischemic stroke in young adults (18-45 years) without classical cardiovascular risk factors represents a diagnostic challenge. In this age group, strokes account for approximately 10% to 15% of all stroke cases [8], with ischemic stroke being the most common subtype (60%-70%). According to the Trial of Org 10172 in Acute Stroke Treatment (TOAST) classification [8], the etiological distribution in young patients is as follows: cardioembolism (21.2%), large artery disease (10.6%), small-vessel disease (9.6%), other determined etiology (19.2%), and undetermined etiology (39.4%). Within this framework, CaW can be classified as another determined etiology [9].

Although CaW is not among the most frequent causes of stroke, it is estimated to account for up to 10% to 20% of cryptogenic strokes [4,10]. Symptomatic CaW represents a high-risk source of cerebral embolism. Observational studies suggest that conservative pharmacotherapy alone may carry a substantial risk of recurrent events [3]. In contrast, interventional treatment, including carotid artery stenting or carotid endarterectomy, is associated with low recurrence rates, although high-quality comparative evidence remains limited [10].

In the differential diagnosis, an ulcerated atherosclerotic plaque was considered but ruled out due to the patient's young age, lack of classical atherosclerotic risk factors, absence of changes in other arteries, and the characteristic location of the lesion within the carotid bulb [8]. Doppler ultrasonography demonstrated a characteristic shelf-like intraluminal projection arising from the posterior wall of the carotid bulb, consistent with CaW. CTA also did not demonstrate typical imaging features of arterial dissection, such as long-segment stenosis, a double lumen, or an intimal flap [11]. Although the morphology suggested CaW, a comprehensive workup was necessary. Echocardiography and Holter ECG ruled out structural cardiac abnormalities and arrhythmias. Given the patient's young age and miscarriages, extended prothrombotic evaluation excluded antiphospholipid syndrome and inherited thrombophilia [12]. The final diagnosis of CaW was based on the characteristic findings on multimodal vascular imaging and was not confirmed by histopathological examination.

Previous methylphenidate use was also considered because it has been associated with an increased risk of cardiovascular events [13]. However, the patient had discontinued the medication more than 6 months before stroke onset, making its contribution to the stroke unlikely.

The most frequently reported diagnostic pitfall is misclassifying CaW as an ulcerated atherosclerotic plaque. In some cases, sagittal CTA reconstruction is essential to reveal its shelf-like morphology. CaW is also frequently overlooked or misdiagnosed as

carotid dissection, carotid bulb thrombus, fibromuscular dysplasia, pseudoaneurysm, or non-calcified plaque [14].

Particular attention should be paid to young patients presenting with recurrent ischemic stroke without an identified etiology [15]. Multimodal vascular imaging plays a central role. CTA is the diagnostic modality of choice to visualize the shelf-like contrast-filling defect along the posterior wall of the carotid bulb [2,16]. Cerebral CTA enables rapid confirmation of stroke and precise vessel localization for mechanical thrombectomy [14], while cervicocerebral CTA reveals the embolic source leading to distal occlusions [17].

Identification of the source facilitates immediate planning and secondary prevention. Duplex ultrasonography combined with CTA provides high diagnostic accuracy [14], while digital subtraction angiography remains the gold standard when interventional treatment is planned [14,18]. As CaW is visible on routinely performed imaging, greater attention should be directed toward recognizing its characteristic features [16,19].

Targeted CTA of the carotid bifurcation is increasingly recommended in young patients with cryptogenic stroke [14]. The optimal management strategy for CaW remains a matter of ongoing debate, as conservative treatment with antiplatelet or anticoagulant therapy alone has been associated with a substantial risk of recurrent embolic events [3]. For example, one reported case involved a patient with ischemic stroke and a lesion within the carotid bulb of the ICA. Despite ongoing anticoagulant therapy, follow-up imaging performed 6 months later revealed a thrombus attached to the lesion, which was subsequently diagnosed as CaW. This finding underscores the limitations of pharmacological intervention in eliminating the underlying thrombogenic focus [20].

In the acute phase, our patient presented with an occlusion of the M2 and M3 segments. As the time window for intravenous thrombolysis had passed, a staged strategy was adopted, starting with mechanical thrombectomy [12]. Achieving complete reperfusion (mTICI 3) was crucial for a favorable outcome [21]. Following thrombectomy, the patient was qualified for angioplasty with carotid artery stenting.

Interventional management of CaW with carotid artery stenting or carotid endarterectomy shows low recurrence rates compared to medical management [3,4,14], which carries a higher risk of recurrent events [3]. Mechanically, carotid artery stenting apposes the CaW against the arterial wall, smoothing the luminal surface and reducing local flow disturbances that favor thrombus formation [1]. While carotid endarterectomy carries a lower risk of periprocedural stroke, and carotid artery stenting carries a lower risk of open surgical complications, young patients show similar outcomes for both procedures.

Consequently, carotid artery stenting is often preferred in young adults due to its minimally invasive nature [22].

Due to the absence of randomized controlled trials and formal guidelines, case reports remain valuable sources of evidence for CaW management. This case illustrates the importance of a comprehensive diagnostic approach in young patients with ischemic stroke. Previously published reports have described CaW as an underrecognized cause of cryptogenic stroke and emphasized its overlap with plaque or dissection [15-18].

The present case is largely confirmatory rather than entirely novel. Its educational value lies in the detailed presentation of the sequential diagnostic process and the resulting management pathway. In particular, it demonstrates how multimodal vascular imaging helped differentiate CaW from competing diagnoses in a young patient without conventional vascular risk factors and guided subsequent treatment decisions, including staged thrombectomy and delayed carotid artery stenting. This case therefore supports increased awareness of CaW during routine stroke evaluation.

Conclusions

CaW should be considered as a potential embolic source in selected young and middle-aged patients presenting with cryptogenic or embolic-pattern ischemic stroke, particularly in the absence of traditional vascular risk factors. This case illustrates that subtle vascular abnormalities within the carotid bulb may represent a clinically relevant thrombogenic lesion and should prompt careful diagnostic evaluation.

Accurate diagnosis may be supported by high-resolution vascular imaging, particularly CTA, which enables identification of the characteristic shelf-like intraluminal projection. In this case, complementary vascular imaging helped distinguish CaW

from other carotid bulb abnormalities and supported subsequent multidisciplinary treatment planning.

The present case also illustrates a feasible diagnostic and interventional pathway. Mechanical thrombectomy was performed in the acute phase of stroke, followed by delayed carotid artery stenting after exclusion of alternative stroke etiologies. However, conclusions regarding recurrence reduction or definitive treatment effectiveness cannot be drawn from a single case report and should be interpreted within the context of the existing literature.

Overall, this report emphasizes that CaW should be considered in young patients with cryptogenic or embolic-pattern stroke. Increased awareness of this condition and appropriate use of advanced vascular imaging may improve etiological diagnosis and facilitate individualized therapeutic decision-making.

Institution Where Work Was Done

Medical University of Łódź, Łódź, Poland

Informed Consent

The patient provided informed consent for the publication of their case details.

AI Disclosure

AI-based language model (ChatGPT) was used for linguistic editing of the text.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

References:

- Chen H, Colasurdo M, Costa M, et al. Carotid webs: A review of pathophysiology, diagnostic findings, and treatment options. *J Neurointerv Surg*. 2024;16(12):1294-98
- Choi PMC, Singh D, Trivedi A, et al. Carotid webs and recurrent ischemic strokes in the era of ct angiography. *Am J Neuroradiol*. 2015;36:2134-39
- Haussen DC, Grossberg JA, Bousslama M, et al. Carotid web (intimal fibromuscular dysplasia) has high stroke recurrence risk and is amenable to stenting. *Stroke*. 2017;48:3134-37
- Zhang AJ, Dhruv P, Choi P, et al. A systematic literature review of patients with carotid web and acute ischemic stroke. *Stroke*. 2018;49:2872-76
- Olindo S, Marnat G, Chausson N, et al. Carotid webs associated with ischemic stroke. Updated general review and research directions. *Rev Neurol (Paris)*. 2021;177:627-38
- Sharashidze V, Nogueira RG, Al-Bayati AR, et al. Carotid web phenotype is uncommonly associated with classic fibromuscular dysplasia: A retrospective observational study. *Stroke*. 2022;53:e33-36
- Boesen ME, Eswaradass PV, Singh D, et al. MR imaging of carotid webs. *Neuroradiology*. 2017;59:361-65
- Smajlović D. Strokes in young adults: Epidemiology and prevention. *Vasc Health Risk Manag*. 2015;11:157-64
- Chatzikonstantinou A, Wolf ME, Hennerici MG. Ischemic stroke in young adults: Classification and risk factors. *J Neurol*. 2012;259:653-59
- Liang S, Qin P, Xie L, et al. The carotid web: Current research status and imaging features. *Front Neurosci*. 2023;17:1104212
- Yaghi S, Engelter S, Del Brutto VJ, et al. Treatment and outcomes of cervical artery dissection in adults: A scientific statement from the American Heart Association. *Stroke*. 2024;55:e91-106
- 2021 Guideline for the prevention of stroke in patients with stroke and transient ischemic attack: A guideline from the American Heart Association/ American Stroke Association | *Stroke* [Internet]. <https://www.ahajournals.org/doi/10.1161/STR.0000000000000375> [Access 15 March 2026]
- Cooper WO, Habel LA, Sox CM, et al. ADHD drugs and serious cardiovascular events in children and young adults. *N Engl J Med*. 2011;365(20):1896-904

14. Mac Grory B, Emmer BJ, Roosendaal SD, et al. Carotid web: An occult mechanism of embolic stroke. *J Neurol Neurosurg Psychiatry*. 2020;91(12):1283-89
15. Kumar GGS, Adiga CP, Goolahally LN, Iyer PP. A case report of carotid web: A hidden fiend of stroke. *Apollo Medicine*. 2022;19:48
16. (PDF) Carotid web: the challenging diagnosis of an under-recognized entity. ResearchGate [Internet]. [Accessed March 15, 2026]
17. Grin EA, Raz E, Shapiro M, et al. Atypical carotid webs: An elusive etiology of ischemic stroke. *World Neurosurg*. 2025;196:123770
18. Moreda NS, de Almeida HA, Grillo VTRDS. Carotid web: A little-known etiology of recurrent stroke – Case report. *J Vasc Bras*. 2025;24:e20240126
19. Park CC, El Sayed R, Risk BB, et al. Carotid webs produce greater hemodynamic disturbances than atherosclerotic disease: A DSA time-density curve study. *J Neurointerv Surg*. 2022;14:729-33
20. Sajedi PI, Gonzalez JN, Cronin CA, et al. Carotid bulb webs as a cause of “cryptogenic” ischemic stroke. *Am J Neuroradiol*. 2017;38(7):1399-404
21. Guidelines for the early management of patients with acute ischemic stroke: 2019 update to the 2018 guidelines for the early management of acute ischemic stroke: A guideline for healthcare professionals from the American Heart Association/American Stroke Association | Stroke [Internet]. <https://www.ahajournals.org/doi/10.1161/STR.0000000000000211> [Accessed March 15, 2026]
22. Konieczna-Brazis M, Brazis P, Switonska M, Migdalski A. Carotid web as a cause of ischemic stroke: Effective treatment with endovascular techniques. *J Clin Med*. 2025;14:2568