VDM Vascular Disease Management

CASE REPORT

Mechanical Thrombectomy for Acute Ischemic Stroke Associated With an Ipsilateral Internal Carotid Artery Web

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Abstract

Carotid web, a shelf-like projection located in the carotid bulb's posterolateral wall, can induce flow turbulence and stasis, promoting clot formation that may lead to embolization within the intracranial circulation, thereby causing ischemic stroke. This case report highlights the clinical trajectory of a previously healthy young man who presented with acute right hemiplegia due to left middle cerebral artery occlusion. The patient underwent successful mechanical thrombectomy, during which an ipsilateral carotid web was identified via digital subtraction angiography. The true prevalence of carotid webs remains uncertain, but they are frequently encountered in young individuals who experience ischemic strokes. Despite conventional treatments such as antiplatelet therapy and anticoagulation, these patients remain at risk for recurrent ischemic events. Current recommendations lean toward interventions such as carotid endarterectomy or carotid stenting as effective management strategies. Further research is warranted to enhance our understanding of carotid webs, optimize diagnostic techniques, and refine treatment protocols.

Introduction

Carotid web, characterized by its distinctive shelf-like intraluminal projection within the internal carotid artery bulb, represents a unique entity that shares features with fibromuscular dysplasia.[1] Despite its significance, carotid web remains an under-recognized etiological factor contributing to ischemic stroke, particularly among young adults.[2] The presence of a carotid web is thought to disrupt hemodynamics within the blood circulation, fostering conditions conducive to stasis and subsequent clot formation. This cascade of events may ultimately culminate in distal embolization and occlusion of larger intracranial vessels.[1] In this context, the current study seeks to shed light on the clinical implications of carotid web-associated ischemic stroke and explore potential therapeutic interventions to mitigate its impact.

Case Presentation

A 42-year-old previously healthy man arrived at the hospital with acute onset right hemiplegia and aphasia. He was brought in approximately 2.5 hours after the commencement of these symptoms. The patient's level of consciousness was altered upon arrival. His heart rate was measured at 80 beats per minute, and his blood pressure was 138/82 mm Hg. A random blood glucose level was recorded at 98 mg/dL.

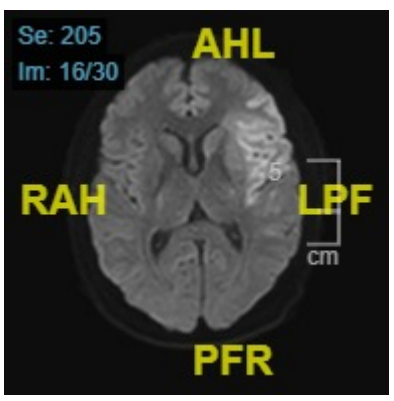

Figure 1. Diffusion-weighted magnetic resonance image showing acute infarct in the left middle cerebral artery territory involving the insular cortex and adjacent frontal and temporal operculum.

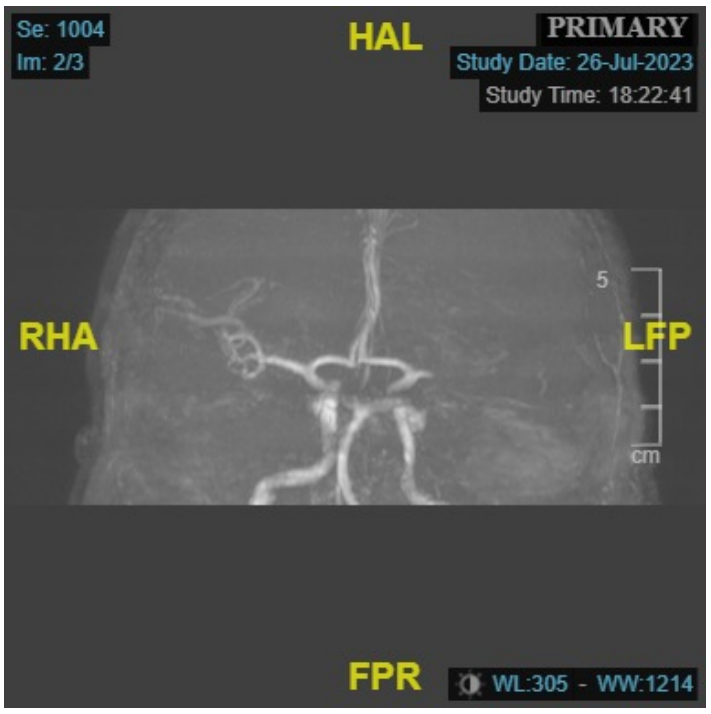

Figure 2. Magnetic resonance angiography maximum intensity projection image showing cutoff of the left M1 middle cerebral artery.

Figure 3. Digital subtraction angiography image of the left carotid injection showing occlusion of the left M1 middle cerebral artery.

Upon physical examination, the patient exhibited a complete lack of strength (0/5) in his right upper limb, and diminished power (1/5) was noted in the right lower limb. His pupils displayed reactivity to light stimulation, and a positive Babinski sign was elicited in the right lower limb. Based on clinical evaluation, he was diagnosed with an acute stroke, and his National Institutes of Health Stroke Scale score was determined to be 16.

Further diagnostic workup included brain magnetic resonance imaging (MRI) and magnetic resonance (MR) angiography of intracranial vessels. The brain MRI revealed evidence of an acute infarct situated within the left middle cerebral artery (MCA) territory, affecting the left insular cortex as well as the adjacent frontal and temporal operculum (**Figure 1)**. Additionally, the MR angiography depicted a cutoff in the M1 segment of the left MCA (**Figure 2)**.

Treatment

Promptly after admission, the patient received immediate treatment through intravenous thrombolysis with a dosage of 30 mg of tenecteplase. Subsequently, a mechanical thrombectomy procedure was conducted, employing a RED 72 reperfusion catheter (Penumbra). Following 2 passes of the catheter, a resilient clot was successfully retrieved. This intervention resulted in the complete recanalization of the MCA, leading to full perfusion of the entire left MCA territory (modified treatment in cerebral infarction score of 3, a successful recanalization). Notably, during digital subtraction angiography, an incidental finding revealed a shelf-like filling defect within the left carotid bulb, which was subsequently identified as a carotid web (**Figures 3, 4**, and **5**).

Following the procedures, the patient was closely monitored in the intensive care unit for 24 hours. Subsequently, he was transferred to a general ward setting. A computed tomography (CT) scan of the brain performed 24 hours after the interventions indicated no evidence of intracranial hemorrhage. The patient's treatment regimen was augmented with 75 mg of aspirin.

Outcome and Follow-up

The patient demonstrated a positive trajectory during his hospitalization, ultimately leading to his discharge after 3 days in stable condition. At the time of discharge, the patient exhibited improved strength, attaining a power of 4/5 in both the right upper limb and lower limb; however, aphasia persisted. To further investigate potential contributing factors, a comprehensive evaluation was

conducted, including 2D echocardiography and a prothrombotic workup, both of which yielded unremarkable findings. To address the carotid web, the patient's management plan was designed to incorporate left carotid stenting, with the procedure scheduled for 2 weeks following his discharge.

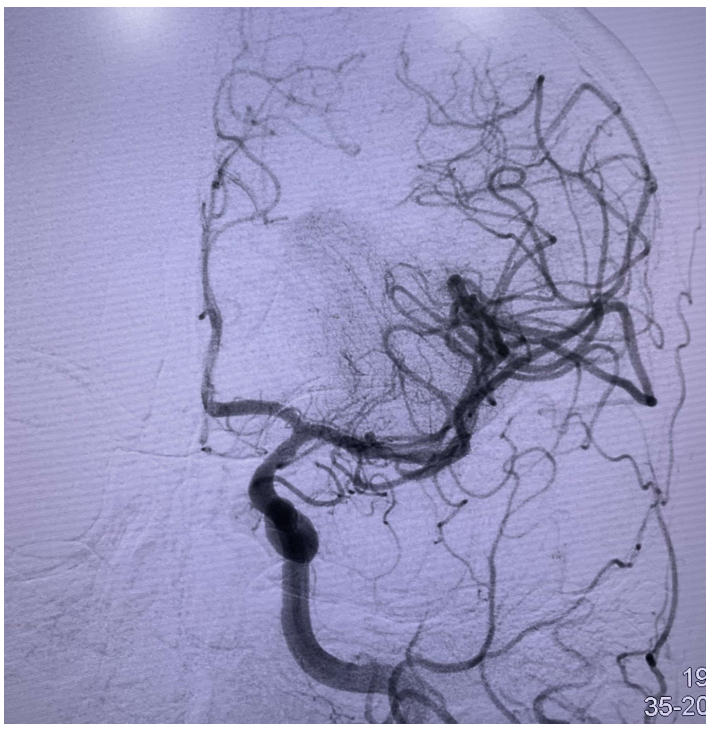

Figure 4. Digital subtraction angiography image of the left internal carotid artery after aspiration thrombectomy showing complete revascularisation of the left middle cerebral artery.

Discussion

The recognition and understanding of a carotid web and its association with ischemic stroke date back to its initial description in 1968.[3] Considered an atypical variant of fibromuscular dysplasia, the precise prevalence of carotid webs remains elusive; however, recent investigations highlight their significant role in ischemic strokes among young adults.[4,5] The development of turbulence and stasis within the arterial lumen, proximal to the carotid web, fosters a milieu conducive to thrombus formation and stagnation. The resultant thrombus can give rise to artery-to-artery embolic strokes, particularly if the thrombus attains a considerable size.[1] In the present case, the absence of other stroke risk factors suggests that the stroke event was likely triggered by embolization from the thrombus stemming from the carotid web.

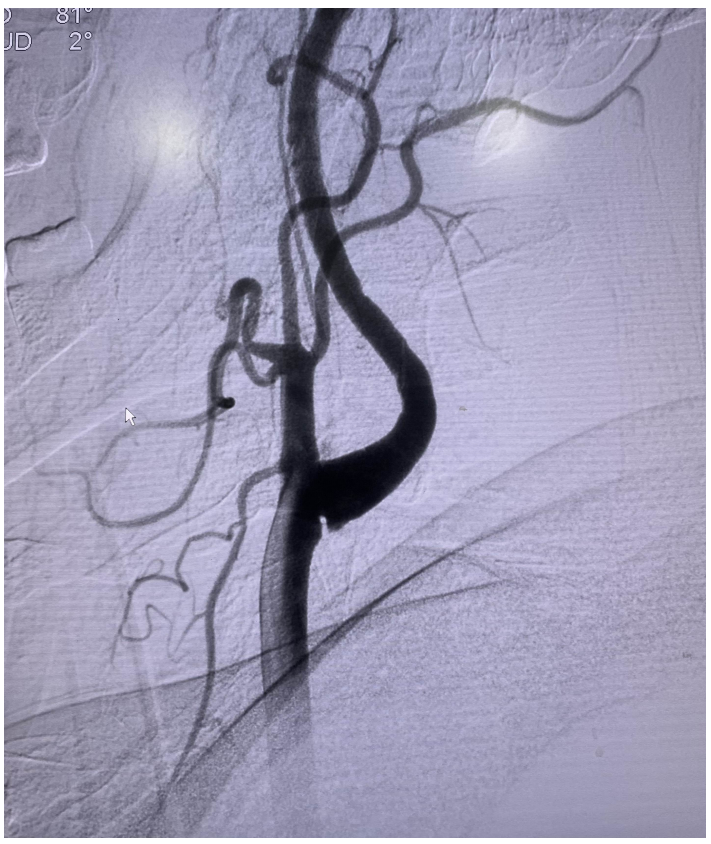

Figure 5. Digital subtraction angiography image of the left carotid bifurcation showing an intraluminal shelf-like filling defect in the left carotid bulb—the "web."

Carotid webs can manifest as either unilateral or bilateral anomalies. In a study by Haussen et al, it was found that 58% of patients with carotid webs exhibited bilateral involvement.[6] While various imaging modalities such as color Doppler imaging, CT angiography, and MR angiography can aid in the diagnosis of carotid webs; a heightened degree of suspicion during image interpretation is crucial to avoid overlooking these subtle abnormalities. Digital subtraction angiography, characterized by its superior temporal and spatial resolution, is considered the gold standard for diagnosing carotid webs.[1]

As of now, treatment guidelines for carotid web-related conditions remain unsettled due to the paucity of well-established clinical trials. Conventional therapeutic approaches involve the use of antiplatelets, statins, and anticoagulants. However, emerging evidence suggests that conservative management of symptomatic carotid webs could leave patients at a heightened risk for future ischemic events.[6] Recent studies lean toward interventions such as carotid endarterectomy or carotid stenting for managing symptomatic carotid webs.[7,8] In alignment with this trend, our case subject was slated for carotid stenting at a later juncture.

Conclusion

Carotid web represents an under-recognized etiological factor for stroke among young adults. Vigilance in the assessment of angiographic images is imperative in cases of stroke occurring in the young population. Although treatment guidelines for carotid web-related strokes are still evolving, emerging evidence favors interventions such as carotid endarterectomy or stenting for addressing symptomatic carotid webs. ■

The authors have completed and returned the ICMJE Form for Disclosure of Potential Conflicts of Interest. The authors report no financial relationships or conflicts of interest regarding the content herein.

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