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IMAGE

Carotid web as a cause of embolic stroke of undetermined source in a young woman

Banda carotídea como causa de infarto cerebral embólico de origen indeterminado en una mujer joven

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A 42-year-old female patient with an unremarkable past medical history sought emergency medical care due to dysarthria, altered sensitivity, and weakness in her left limbs. Intravenous thrombolysis could not be offered, and thrombectomy was not available in our center. When she was admitted, she had stable vital signs, and the physical examination showed left facial hypoesthesia and dysesthesia. The proximal and distal weakness were 2/5 and 0/5 respectively on the MRC scale. The initial cranial tomography ruled out hemorrhage; but the diffusion-weighted MRI showed an infarction in the right frontal and temporal lobes, also affecting the basal ganglia (Fig. 1A). A transthoracic and contrast transesophageal echocardiogram did not show any heart abnormalities. The Holter ECG and the carotid doppler were also normal. Subsequently, a computed tomography angiography (CTA) demonstrated a thin intraluminal filling defect along the posterior wall of the right carotid bulb (Fig. 1B), which appeared as a septum on axial imaging (Fig. 1C). From a posterolateral view, the 3D reconstruction CTA showed the web as an indentation of the carotid wall (Fig. 1D). No other abnormalities were found after extensive diagnostic tests, including infectious and autoimmune etiologies and acquired/hereditary thrombophilia. The patient and her family refused any type of endovascular or endarterectomy intervention. Two year later, the patient continues her rehabilitation and her antithrombotic therapy with acetylsalicylic acid (ASA), without stroke recurrence. The patient gave her informed consent to publish her case.

The carotid web (CW) is thought to represent an intimal variant of fibromuscular dysplasia since it was first described by Rainer et al. in 1968.[1] A close linkage between focal fibroplasia or hyperplasia within the innermost layer of the arterial wall has been noted. It has been considered that a significant abnormal flow in the CW with turbulence and stasis could create a thrombogenic milieu with a subsequent embolization of fibrin-based clots.[2] Usually, the CW is located from the dorsal aspect of the internal carotid artery just beyond the bifurcation. It has a smooth contour, and it is associated with a normal bulb shape. To differentiate the CW from arterial dissection, it is important to consider that the dissection typically affects the middle or distal cervical segments of the internal carotid, imaging commonly shows irregular borders, and it may involve an enlargement of the arterial diameter related to the false lumen (pseudoa-

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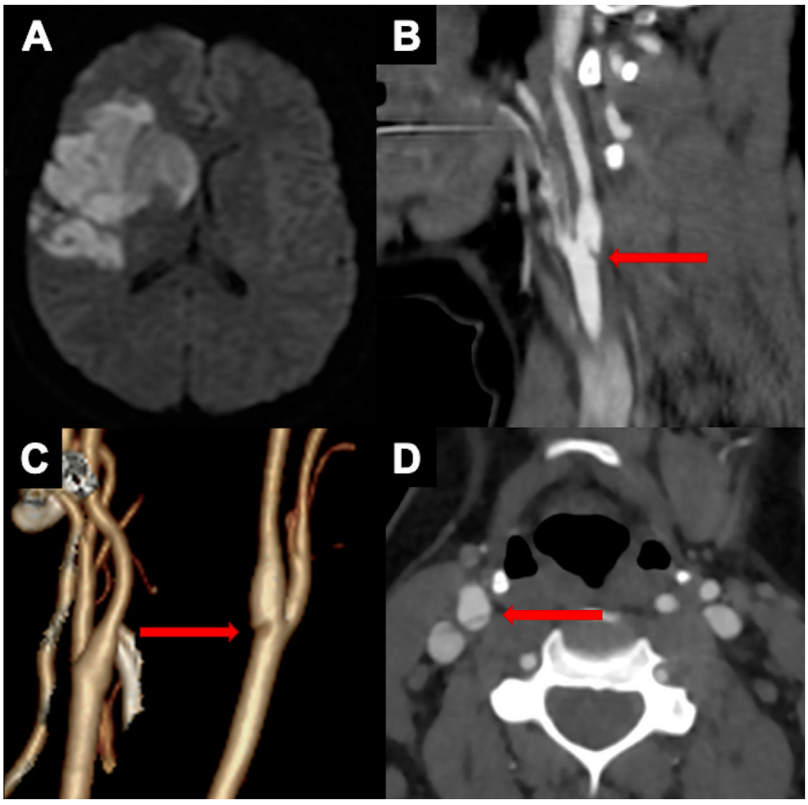

Figure 1 (A) Diffusion-weighted magnetic resonance imaging shows an infarction in the frontal and temporal lobes, also affecting the basal ganglia. (B and D) Computed tomography angiography demonstrates a thin intraluminal filling defect (arrow) along the posterior wall of the right carotid bulb which appeared as a septum on axial image. (C) A postero-lateral view of the 3D reconstruction computed tomography angiography shows the web like an indentation of the carotid wall.

neurysm). CW may be misinterpreted because they do not cause haemodynamically significant stenosis and may mimic an arterial dissection, a non-calcified atherosclerotic plaque or an intraluminal thrombus.[3]

The prevalence of CW has not been conclusively determined; some registries show data between 1.2 and 2.7%, but case-control series that examined patients under 60 years of age demonstrated 9.4–37% prevalence of a CW ipsilateral to the stroke.[3] In a systematic review including 158 patients with a median age of 46 years (range 16–85), 57% did not have stroke risk factors, and 56% had recurrent stroke with a median of 12 months (range 0–97) despite the use of antithrombotic therapy.[4] It has been reported that patients who received carotid revascularization (stenting or endarterectomy) have not had recurrence during a 24 months follow-up (range 13–35).[5] To date (2 years later) our patient has exceeded expectations and is part of the non-recurrence group despite not having received endovascular therapy or anticoagulation.

The CW is not an uncommon cause of stroke in young people without other risk factors, especially when an embolic stroke of undetermined source is suspected. Because there is a high risk of stroke recurrence, a carotid revascularization should be offered to patients. Regarding antithrombotic therapy, the standard of care continues to be ASA.

Authors' contributions

Conceptualization: Cristian Eduardo Navarro; Data curation: Jonatan Álvarez-Ortega; Formal Analysis: Cristian Eduardo Navarro, Jonatan Álvarez-Ortega; Investigation: Cristian Eduardo Navarro, Jonatan Álvarez-Ortega; Methodology: Cristian Eduardo Navarro; Project administration: Cristian Eduardo Navarro; Resources: Cristian Eduardo Navarro; Software: Cristian Eduardo Navarro; Supervision: Cristian Eduardo Navarro; Validation: Cristian Eduardo Navarro; Visualization: Cristian Eduardo Navarro; Writing – original draft: Jonatan Álvarez-Ortega; Writing – review & editing: Cristian Eduardo Navarro.

Ethical approval

Ethics approval is not required for image publication at our institution.

Consent to participate and consent to publish

The patient gave her informed consent to participate in this case and to publish the image attached.

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Conflicts of interest

The author declares no competing interests.

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