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Acute Ischemic Stroke in a 41-year-old Female Secondary to Internal Carotid Artery Dissection: A Case Report

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ABSTRACT

Spontaneous carotid artery dissection is an important cause of ischemic stroke and is most common in young and middle-aged adult patients. Dissection occurs after tearing of the intima layer of the artery causes the formation of a thrombus (intramural), and it leads to thrombosis or embolism. The thrombosis or embolism, in turn, interrupts arterial circulation and, subsequently, induces infarction in the territory of the affected artery. The case of an ischemic vascular event involving the left cerebral hemisphere secondary to a spontaneous dissection of the left internal carotid artery is presented here.

KEYWORDS: acute ischemic stroke, carotid artery dissection, thrombolysis, angioresonance

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INTRODUCTION

Stroke is one of the leading causes of death and disability worldwide. It is generally classified as ischemic or hemorrhagic. The occlusion of an artery is the most common cause of ischemic stroke, being responsible for 71% of all strokes worldwide.[1,2] The main unmodifiable risk factors for ischemic stroke are gender, genetics, and age, with the likelihood increasing after 39 years of age in developing countries while arterial hypertension is the main modifiable risk factor.[1] Dissection in the intimate layer of an artery with formation of intramural thrombus is an important cause in young patients, most occur in the carotid and extra cranial vertebral artery occluding the artery at the site of dissection or formation of thrombosis or distal embolism.[1,2] Dissection is often associated with exertion (e.g., trauma or coughing) but may occur spontaneously.[1]

The clinical presentation of an ischemic stroke is the sudden appearance of a neurological deficit originating in the territory of the occluded artery. Such deficits are classified as mild to very severe assessed on the NIHSS scale.[2] Routinely based on AHA algorithms, hypoglycemia and intracerebral hemorrhage should be ruled out.[2,3] The study of choice to rule out bleeding is tomography (CT) of the skull without contrast because this type of imaging is highly sensitive and widely available in medical centers.[2] In typical cases, a tomographic study is sufficient to diagnose acute ischemic stroke, with a sensitivity of almost 100% in detecting intracranial hemorrhage. Magnetic resonance imaging (MRI), which features special sequences for the evaluation of the structure and function of brain tissue, is the most sensitive imaging study for the detection of acute ischemia.[1,2] The in-hospital treatment of ischemic stroke depends on the time of onset. For instance, in patients with an NIHSS scale between 5 and 25 points, thrombolytic therapy with alteplase is recommended within 3 to 4.5 hours after onset at a dose of 0.9 mg per kilogram of body weight and a maximum of 90 mg, with 10% of the initial bolus dose administered over 1 minute and the rest over 1 hour.[3] For an acute ischemic stroke, endovascular therapy is indicated from 4.5 to 12 hours after onset but is contraindicated when the cerebral infarction is large.[4]

CLINICAL CASE

This case report focuses on a 41-year-old woman, a housewife residing in Zacatecas, Mexico, with no significant medical history. She presented to the emergency department

with severe holocranial headache, nausea, aphasia, and weakness of the right hemibody, all of recent onset. Her vital signs were blood pressure 90/50 mmHg, heart rate 54 bpm, respiratory rate 21 rpm, and oxygen saturation 87%. In the physical examination with global aphasia and right hemiplegia, capillary glycemia reported in 112 mg/dL is performed on the skull tomography without evidence of hemorrhagic lesions, is classified as ischemic stroke with a 19-point NIHSS initiating thrombolysis at 3 hours with alteplase 67 mg, obtaining a 24-hour NIHSS score of 16. As part of the diagnostic protocol, an electrocardiogram was requested, which was normal (i.e., without evidence of arrhythmia). In addition, Doppler ultrasound of both carotids (Figure 1) revealed an atheroma plaque with hemorrhage in the intima proximal to the left carotid bulb and stenosis of 48%.

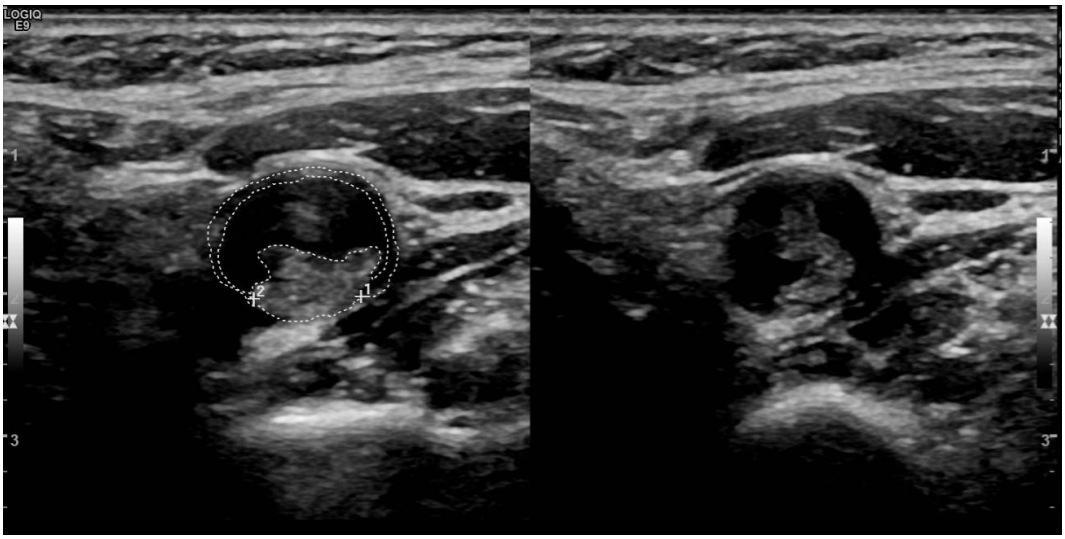

Figure 1. Left carotid artery ultrasound showing atheroma plaque producing 48% stenosis of the blood vessel.

MRI of the brain was requested (Figure 2) and showed the presence of an ischemic area in the territory of the left internal carotid artery, specifically, in the angioresonance of the supra-aortic trunks, with common carotid stenosis proximal to the bifurcation (Figure 3), thus suggesting carotid dissection. The patient's total cholesterol was 171 mg/dL (normal 140-220 mg/dL), with triglycerides 145 mg/dL (normal 6-200 mg/dL), very low-density cholesterol (VLDL) 29 mg/dL (normal 10-48 mg/dL), high-density cholesterol (HDL) 43 mg/dL (normal 45-65 mg/dL), and low-density cholesterol (LDL) 141 mg/dL (normal 100-190 mg/dL).

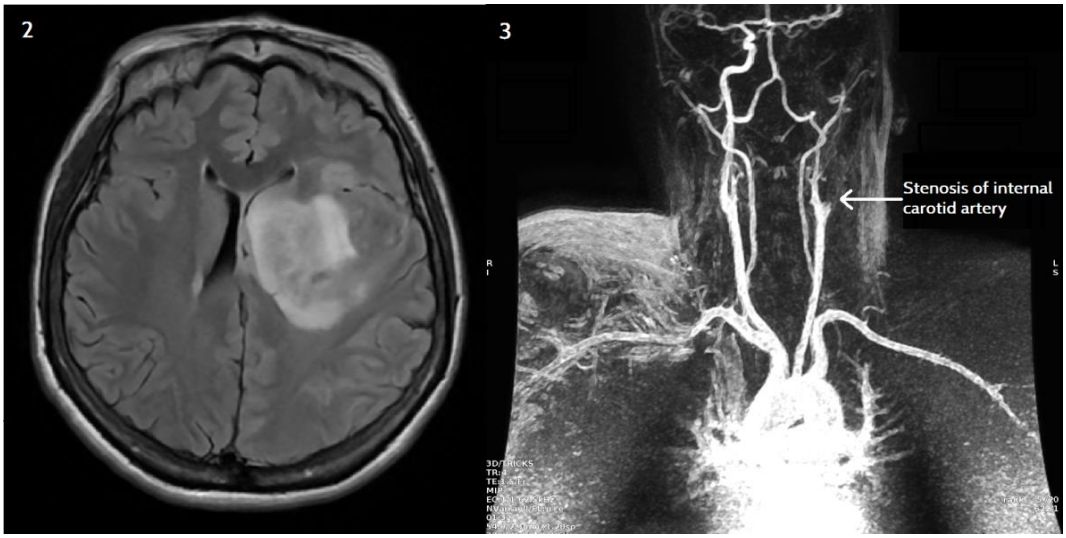

Figure 2. Brain magnetic resonance in T2 FLAIR sequence showing a change of intensity (hypointense) of the left cerebral hemisphere with perilesional edema compressing the cerebral ventricles. Figure 3. Angioresonance showing area of stenosis 1 cm from the left carotid bifurcation.

CONCLUSION

Arterial dissection occurs most frequently in the intracranial or extracranial segments of the carotid and vertebral arteries. The involvement of these neck arteries increases the risk of stroke or transient ischemic attack. The dissection usually results from a tear in the intima that causes the formation of

intramural thrombus and produces, in turn, stenosis of artery, while spontaneous dissection can result from a weakness in the arterial wall. The clinical manifestations of internal carotid artery involvement are unilateral headache (the most frequent symptom), neck pain, anisocoria, paresthesia, visual symptoms or Horner syndrome, and late cerebral or retinal ischemia. Twenty percent of patients with spontaneous dissection suffering ischemic stroke are asymptomatic. Angiography is the gold standard for the diagnosis of arterial dissection, with stenosis typical of the involved artery. Intimal flap or double light occurs in only 10% of patients. Treatment is based on preventing the complications (thrombotic or embolic) with heparin and oral warfarin for patients with acute carotid or vertebral artery dissection. Endovascular or surgical treatment is reserved for patients with persistent ischemia despite anticoagulant treatment since arterial dissection most often cures spontaneously.[6,7,8] In this patient, a diagnosis of internal carotid aortic dissection was made, and outpatient treatment with HMG-CoA reductase inhibitor (atorvastatin 80 mg/day) and oral anticoagulant (rivaroxaban 15 mg/day) was initiated in collaboration with physical rehabilitation medicine.

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