Case Report

Headache and Spontaneous Internal Carotid Artery Dissection

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Introduction

Dissection of the extracranial carotid arteries is increasingly recognized and is a common cause of stroke in young patients. In the absence of a traumatic trigger, spontaneous internal carotid artery dissection (CAD) may be related to various well-defined pathological processes.[1] It is likely to be multifactorial, including environmental exposures, infections, and nonspecific inflammation of the arterial wall.[1] A wide spectrum of sports and different physical activities has been reported to precede spontaneous CAD.[6,7] However, most frequently, the etiology remains undefined as in the present case.

Case Report

A 32-year-old female was brought by her friend to the emergency room (ER) after she had severe headache and near-syncope event at her work place. As per her friend, the patient's headache started the night before; it was gradually getting worse to this point. In the ER, she became more lethargic and was noted to have left facial droop. Immediately, she developed respiratory distress and was intubated.

Patient's histories were systematically screened and ruled out migraine, trauma, and infections during the two weeks before the headache onset. No fever (temperature≥38°C) or any other typical symptoms (cough, rhinitis, hoarseness, sneezing, vomiting) with onset of the headache.

She has no history of alcohol, cigarettes, and illicit drug use. She had bilateral ovarian tube ligation and no use of contraceptive pills.

The patient was sedated. Blood pressure was 120/70 mmHg, heart rate 65 beats/minute, oxygen saturation 100% on AC-Ventilator (500/12/70), and temperature 37° C (98.6° F). There were no heart murmurs, rubs, or gallops. There were no carotid bruits. Right-side weakness was accompanied by decreased sensation and positive Babinski sign.

Results of basic metabolic panel, CBC, cardiac enzymes, and coagulation panel were normal. Urine analysis and urine drug screen were negative for infection or illicit drug. CT scan of the brain revealed large left middle cerebral artery (MCA) territory acute infarct but no bleeding.

The patient was admitted to the Intensive Care Unit (ICU) for respiratory failure secondary to acute stroke. Neurology and neurosurgery were consulted. Aspirin was given. An MRI of the brain done 24 hours later confirmed the result of brain CT and expected progression (Figure 1). MR angiography of brain and neck revealed patent left middle cerebral artery (Figure 2) but severe dissection at the left carotid bulb (Figure 3). According to the neurosurgeon, any attempt toward intervention at revascularization of the left internal carotid artery was not encouraged because of the risk of hemorrhagic conversion with regard to the stroke. Given the large size of the infarct, the neurosurgeon recommended continuing low-dose aspirin and subcutaneous heparin 5,000 units every eight hours for deep venous thrombosis (DVT) prophylaxis.

To determine the cause of the stroke, a transthoracic echocardiogram was performed and shown negative for the possibility of endocarditis, with no cardiac cause for an embolism. The other results, including hypercoagulopathy panel, rheumatoid

factor, lipid profile, C-reactive protein (CRP), anti-nuclear antibody (ANA) and ESR, were all within normal limits.

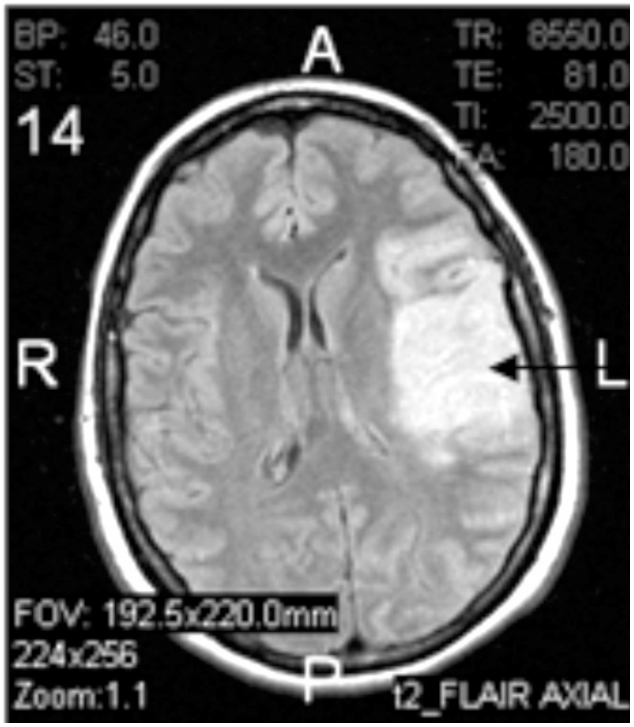

Figure 1. MRI of the brain revealed large left middle cerebral artery (MCA) territory acute infarct, as indicated by the arrow.

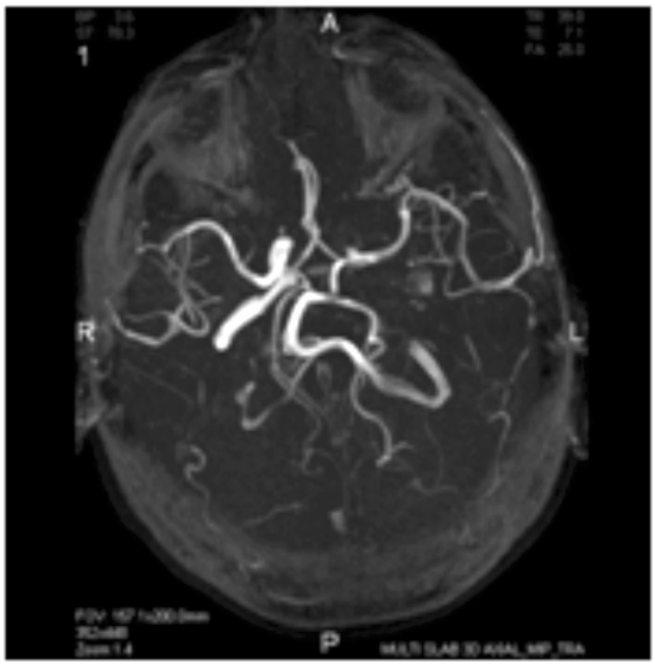

Figure 2. MR angiography of brain revealed left middle cerebral artery was patent.

Serial CT scans showed stabilized infarct and reduced edema. Patient was extubated on third day of her hospitalization. At discharge, the patient was switched to a full dose of aspirin and Plavix for four to six weeks and then aspirin for rest of her life time. Patient was transferred to acute rehabilitation center.

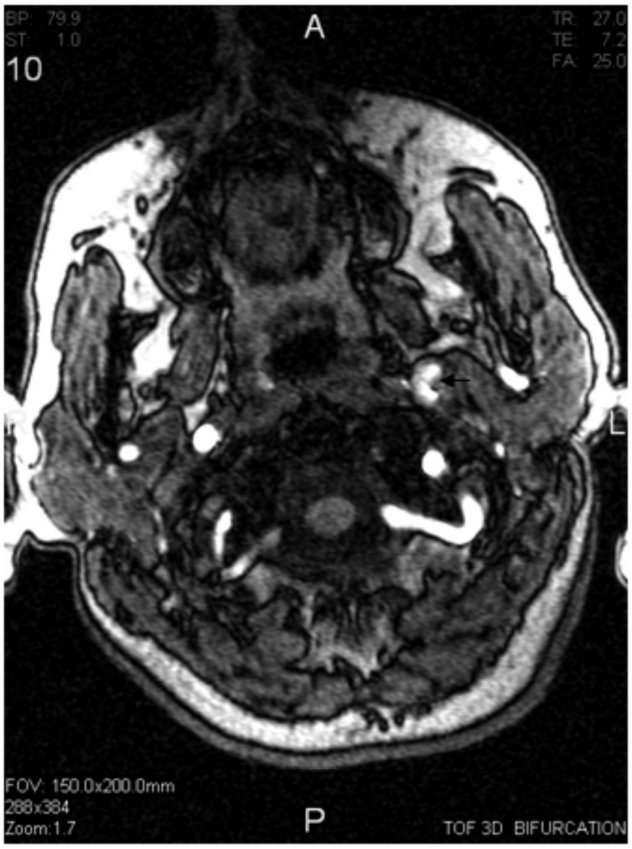

Figure 3. MR angiography revealed severe dissection at the left carotid bulb. Please note the bleeding interrupted lumen indicated by the arrow.

Discussion

Naggara et al[4] studied 29 consecutive patients with CAD by high resolution of MRI. They reported that symptomatic spontaneous internal CAD was more frequently associated with the presence of periartery soft tissue edema compared with traumatic CAD. Their study and the others[5,6,7] also indicated that biological markers of inflammation are significantly correlated with the imaging finding in patients with spontaneous internal CAD.

According to the history, the patient in this case had no history of neck injury, any provoking physical activities, or sickness before this event. Although high resolution MRI was not performed during hospitalization, the inflammatory biomarkers were negative. In agreement with Naggara's group, in order to define the etiology in a larger population of CAD, we encourage the use of high resolution MRI to explore vasculopathies other than atherosclerosis. Further studies with more specific proinflammatory markers related to systemic vasculitis; i.e., IL-6 and IF-g, are warranted to corroborate the role of inflammation in spontaneous CAD.

The principal mechanism of carotid artery dissection is thromboembolism.[8] Endovascular repair and surgery are justified as an alternative therapeutic approach to achieve a sufficient restoration of the vessel lumen. In a nonrandomized study,[9] the data suggested that frequency of new cerebral ischemic events

in patients with spontaneous CAD is low and probably independent of the type of antithrombotic treatment (aspirin or anticoagulants). Given the unfavorable results of surgery associated with the possible hemorrhagic conversion of the stroke in the present case, we started antithrombotic treatment with aspirin and Plavix.

Conclusion

CAD accounts for up to 10% of ischemic stroke events in young adults. Although the long-term recurrence rate of symptomatic dissection is low, patients should be informed about warning symptoms of dissection and to obtain screening vascular imaging. Failure to make a precise early diagnosis and start appropriate treatment may result in long-term neurologic sequelae or death. Based upon the mechanism of the disease, antithrombotic treatment with anticoagulatives or aspirin remains the first-line treatment and disease control.

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