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Case Report

A Young Woman with Sudden Hemiparesis

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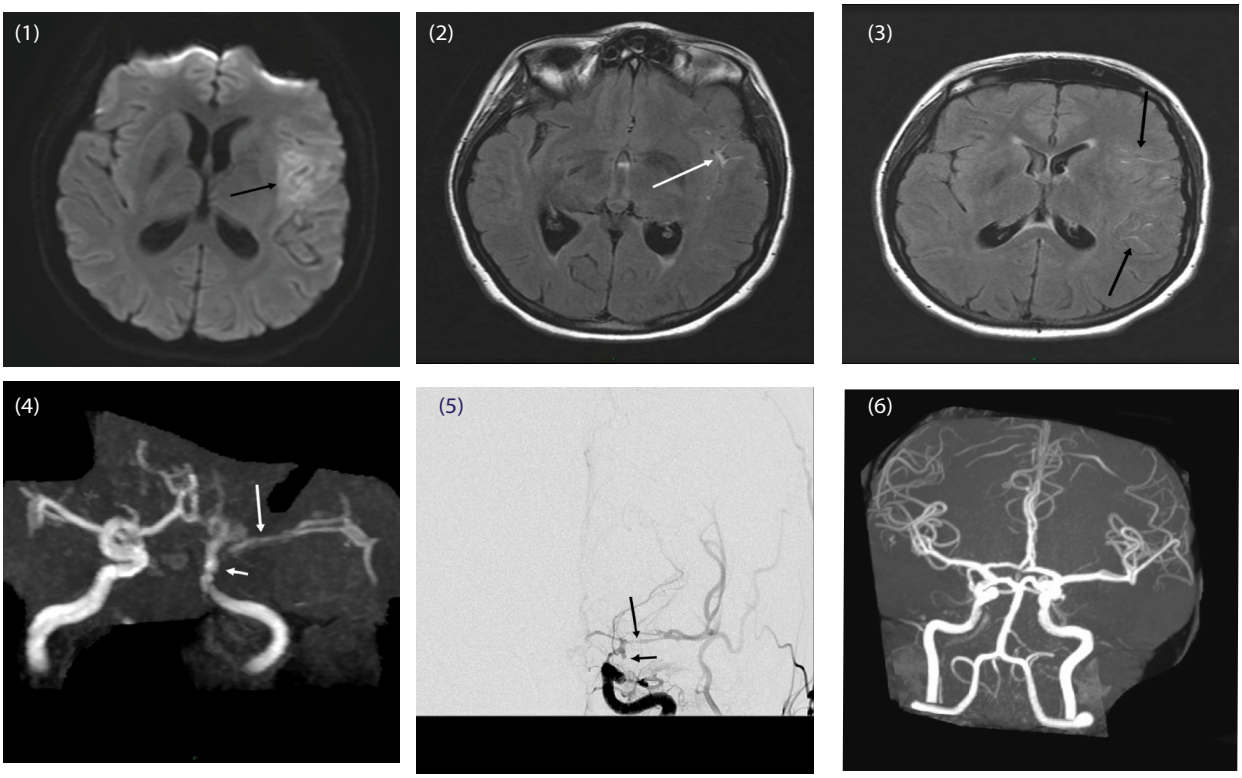

Figure 1: Diffusion weighted imaging (DWI) shows hyperintensity indicative of core infarction on MRI within less than 3 h after onset.

Figure 2: Fluid-attenuated inversion recuperation imaging-Hyperintense vessels proximal to the sylvian fissure.

Figure 3: Fluid-attenuated inversion recuperation imaging-Hyperintense vessels distal to the sylvian fissure.

Figure 4: Magnetic resonance angiography-Severe vasospasm of the left internal carotid artery (short arrow) and middle cerebral artery (M1 segment) (long arrow).

Figure 5: Digital subtraction angiography-Severe vasospasm (long arrow) and small aneurysm of the left anterior choroidal artery of 10 mm of diameter (short arrow).

Figure 6: Magnetic resonance angiography follow-up-Exclusion of the aneurysm and normal caliber of the cerebral arteries.

Abstract

Stroke related to cerebral vasospasm may have the same appearance that ischemic stroke of other causes. The correct diagnosis is critical to avoid inappropriate treatments as thombolysis and anticoaglant therapy. In this case, a previous young healthy woman presented with acute onset of right-sided hemiplegia and expressive aphasia. Interpretation of Magnetic Resonance Images was challenging: acute ischemia, severe vasospasm, no signs of subarachnoid hemorrhage or intra-arterial thrombi, no evidence of cervical artery dissection. Cerebral angiography allowed to conclude that a ruptured small aneurysm was responsible for stroke, related to a spasm at the 7th day of aneurysm rupture concomitant of neck pain. The predictive MRI signs and the factors that contributed for the good outcome of a symptomatic vasospasm are discussed.

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Case Presentation

A 30-year-old woman, without risk factors, presented with acute onset of right-sided deficit and expressive aphasia for potential thrombolysis.The interpretation of MRI was challenging. Various MRI findings help determine the etiology of stroke. A previously healthy 30-year-old woman presented to the stroke center for potential thrombolysis. She had presented sharp neck pain one week ago and neurological examination was unremarkable. She awoke at 8 am with right motor deficit. On arrival, 4 hours later, she was mutic with a Glasgow Coma Scale score of 15. She was normotensive (110/75 mmHg) with right-sided hemiplegia. Electrocardiogram did not demonstrate any arrthymia or other abnormality

Emergent magnetic resonance imaging (MRI) of the brain displayed on diffusion-weighted imaging MRI (DWI) (Figure 1) and apparent coefficient diffusion map, (within 4 hours from onset), an acute ischemic stroke in the superficial territory of left middle cerebral artery (MCA), with normal fluid attenuated inversion recovery (FLAIR) except vessel hyperintensities (FVHs) proximal and distal to the sylvian fissure (Figures 2 and 3). T2* did not reveal any thrombus or hemorrhage. The presence of hyperintensity on DWI without corresponding hyperintensity on FLAIR, called “DWI-FLAIR mismatch indicate that the patient is likely within the time window for thrombolysis, Magnetic resonance angiography (MRA) visualized a severe vasospasm of the left terminal internal carotid artery and left MCA (M1 segment) (Figure 4). Intracranial and extracranial MRA was otherwise unremarkable, especially no dissection of the left carotid artery was identified, as also demonstrated by three-dimensional black blood (3D CUBE) T1 sequence.

Diagnosis

Aneurysm rupture presenting as delayed ischemic stroke related to severe vasospasm

Discussion

The interpretation of this imaging is challenging. Various MRI findings help determine the etiology of stroke and patient selection for acute ischemic stroke treatment (thrombolysis) [1]. The most common mechanisms of stroke in young population are cardioembolism and patent foramen ovale, and dissection of the cervical arteries. The first cause can be suspected when patients present acute multiple territorial lesions or a single large cortical and subcortical lesion on DWI and patent foramen ovale, another source of cardioembolism, usually causes small cortical lesions in vertebro-basilar territory. The presence of hypointense signals with blooming artifacts in the arterial system on T2* sequence (susceptible vessel sign) assess the presence of intra-arterial thrombi, usually associated with cardioembolic stroke. In this unusual presentation of stroke in a young patient without any risk factor, the absence of visualization of intra-arterial thrombus on T2* sequence and of carotid artery dissection (mural hematoma) on dedicated 3D CUBE sequence and the presence of a long spasm allow to conclude that a ruptured aneurysm was responsible for stroke, related to the spasm that occurred at the 7th day of rupture contemporary of neck pain.

Seven days after the aneurysm rupture, a subarachnoid hemorrhage could not be identified on CT, nor on FLAIR and T2* MRI sequences : even if FLAIR sequence is more sensitive than CT and over a longer time period, the patient underwent MRI to late for examination at the 7th day [2]. Cerebral angiography identified a small aneurysm of the left anterior choroidal artery (Figure 5). Patients with DWI-FLAIR mismatch are likely to be within the time of adequate treatment. Endovascular treatment was performed using mechanical angioplasty and coiling. At 3-month follow-up, the patient was asymptomatic and MRA was normal (Figure 6). FHVs are imaging markers of both occlusion or narrowing when proximal to the ischemic core and of collateral system when distal. When present outside the ischemic core, they are associated with good outcome [3]. In the present case, the presence of collateral flow objective by FHV, an early treatment and the absence of risk factors contributed for the good outcome of a symptomatic vasospasm [4,5].

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