Case Report

Clinical and Radiological Difficulties to Detect Isolated MCA Dissection before Intravenous tPA Therapy

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A 64-year-old woman was admitted to our hospital 48 minutes after sudden onset of dysphasia and right hemiplegia. Head computed tomography revealed small infarcts in the left putamen and 4-dimensional computed tomography angiography depicted high-degree stenosis in the left middle cerebral artery and delayed filling of the contrast media in the left middle cerebral artery territory. The patient underwent intravenous tissue plasminogen activator treatment. On day 5 of hospitalization, the patient underwent conventional cerebral angiography, revealing internal carotid artery to middle cerebral artery dissection. Fortunately, subarachnoid hemorrhage as an adverse effect did not occur, although iv-tPA was administered without detecting middle cerebral artery dissection.

Key Words: Intravenous recombinant tissue plasminogen activator—intracranial artery dissection—ischemic stroke—anterior circulation stroke

Case

A 64-year-old woman was admitted to our emergency room 48 minutes after sudden onset of dysphasia and right hemiplegia (NIH Stroke Scale/Score, 7) with no complaint of headache.

Head computed tomography (CT) revealed hypodense regions in the left putamen but no hemorrhage. A 4-dimensional CT angiography (4D-CTA)[1] demonstrated a clear delayed contrast-enhancement effect and a high-degree stenosis originating in the M2 frontal lobe section but no stenosis originating in the M2 parietal lobe section of the left middle cerebral artery (MCA; Fig 1, A,B). Accordingly, the patient was diagnosed with acute-phase cerebral infarction and initiated intravenous tissue plasminogen activator (iv-tPA) at 102 minutes after onset. Digital subtraction angiography conducted on day 5 of hospitalization revealed an intimal flap continuing from the left supraclinoid internal carotid artery to the M2 segment of the MCA (Fig 1, C). These findings indicated that cerebral infarction occurred due to extensive left MCA dissection. Fortunately, subarachnoid hemorrhage as an adverse effect did not occur, although iv-tPA was administered without detecting dissection. The present study was approved by the Ethics Committee at Shonan Kamakura General Hospital, Kamakura, Japan.

Discussion

Intravenous tPA is effective for cerebral infarction within 4.5 hours of onset; therefore, it must be administered within the target time at the earliest.[2] Although intracranial cerebral artery occlusion may occur due to arterial dissection in some cases,[3] its recognition and diagnosis in the emergency room may be challenging. Notably, tPA may cause subarachnoid hemorrhage in cases of intracranial cerebral artery dissection.[4] Although not completely contraindicated,[5] tPA therapy can be avoided if intracranial cerebral artery dissection could be diagnosed beforehand. Therefore, MCA dissection must always be considered before iv-tPA administration. In the present case, despite 4D-CTA, it was clinically and radiologically difficult to detect MCA dissection before iv-tPA therapy.

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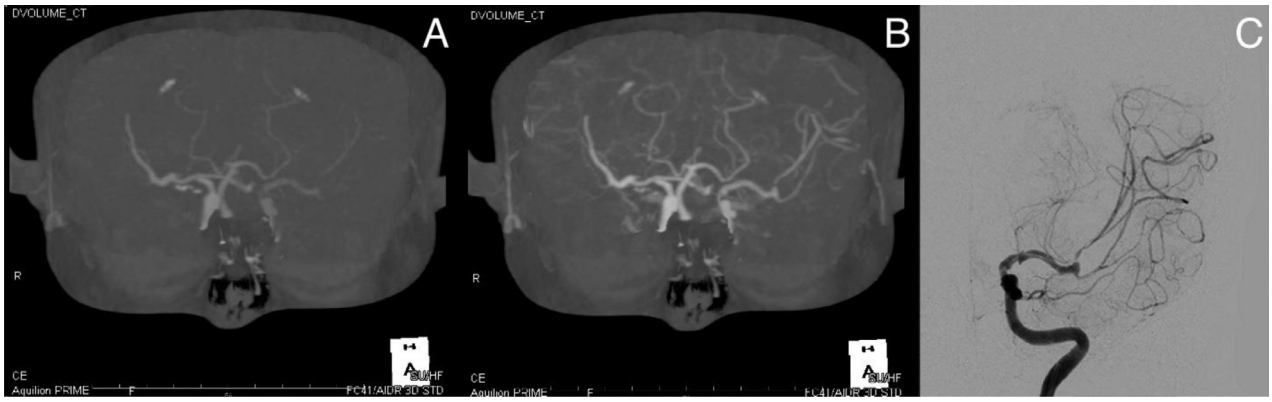

Figure 1. *(A) (left): 4-dimensional computed tomography angiography (4D-CTA) frontal image at admission. (B) (middle): Frontal image of 4D-CTA slightly later than (A). Uptake in the region extending from the left internal carotid artery to the middle cerebral artery (MCA) is less than that on the right. A high degree of stenosis is noted in the left MCA M2 frontal lobe section. Although no stenosis in the origin of M2 parietal lobe section, there is a clear delay in the imaging. (C) (right): An intimal flap extending from the left intracranial internal carotid artery to the M2 segment of the MCA can be noted. Based on the finding, it was diagnosed as cerebral infarction due to extensive intracranial arterial dissection.*

On retrospective review of the 4D-CTA, whole MCA peripheral branches were depicted, although the filling of contrast media was severely delayed and overall density was low. The 4D-CTA findings, probably owing to MCA dissection, showed differences from typical embolic or thrombotic MCA occlusions.[1]

References

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