



DEBATE

Should we do it again?

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Abstract

Clinical Case: A 46-year-old female, with history of iron deficiency anemia due to hiatal hernia, is taken to the emergency department with symptoms of speech disturbance and right-sided paresis. Stroke protocol was activated. Neurologic examination showed global aphasia, left oculocephalic forced deviation, right-sided hemiparesis and hemihypoesthesia – NIHSS 23. A brain CT showed loss of grey-white matter differentiation at the left lenticular nucleus (ASPECTS 9). Prompt treatment with alteplase (0.9mg/kg) was initiated, 205 minutes after the onset of symptoms. CT angiography showed a terminal left internal carotid artery (ICA) and proximal (M1) middle cerebral artery (MCA) occlusion and urgent mechanical thrombectomy was performed with TICI 2a reperfusion of the left carotid circulation. The patient was subsequently admitted to the stroke unit. Close clinical monitoring showed no neurologic improvement at 24 hours. At this point, a control brain CT showed left basal ganglia infarct, as well as hyperdense left middle cerebral artery and a

carotid/transcranial doppler ultrasound (TCD) revealed persistence of the left ICA thrombus, with no significant hemodynamic effect, as well as high resistance and turbulent flow through the M1-M2 segment of the left MCA. The patient remained neurologically stable (NIHSS 18). On the fourth day, a worsened TCD pattern of the proximal MCA prompted an urgent brain CT angiogram, which confirmed a distal left MCA reocclusion and persistence of left ICA thrombus. Despite not having neurological deterioration, after multidisciplinary decision, the patient underwent DSA and mechanical thrombectomy, successfully removing the internal carotid thrombus but incapable of distal MCA reperfusion. No intracranial hemorrhage or neurological deterioration was noted and NIHSS at discharge was 19. Secondary prevention with single anti-platelet and statin therapy was adopted.

Conclusion: Should this patient have been submitted to a late reperfusion, after ischemic stroke and reocclusion, considering there was no neurologic deterioration?

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