Use of Multimodal MRI and Novel Endovascular Therapies in a Patient Ineligible for Intravenous Tissue Plasminogen Activator

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Background and Purpose—Options are limited for individuals who present to the ED within 3 hours of ischemic stroke onset, but who are deemed ineligible for intravenous thrombolysis. Multimodal MRI has been shown to be of great help in identifying stroke patients with large areas of at risk “penumbral tissue”, who may gain from the use of novel endovascular therapies. We report a patient who twice benefited from this management approach, in the setting of hemorrhagic risk following successive ischemic strokes.

Case Description—The patient is a 78 year old male who experienced acute ischemic stroke on 2 separate occasions 5 months apart, and for whom perceived contraindications to appropriate thrombolytic therapy administration led to the successful use of different endovascular therapies at each encounter. Furthermore, following mechanical clot retrieval during the second encounter, the high intensity signal area noted on the baseline diffusion weighted imaging (DWI) in the posterior circulation territory, was almost completely resolved on the day 7 post-procedure MRI.

Conclusion—To our knowledge, this is the first reported case of reversal of a DWI abnormality in the posterior circulation territory. (Stroke. 2005;36:e77-e79.)

Key Words: embolectomy • magnetic resonance imaging, diffusion weighted • stroke, acute • thrombectomy • thrombolytic therapy • tissue plasminogen activator

We report the case of an individual who experienced acute ischemic stroke on 2 separate occasions 5 months apart, and for whom perceived contraindications to appropriate thrombolytic therapy administration led to the successful use of different endovascular therapies at each encounter. Most saliently, we present the first report of diffusion-weighted imaging (DWI) reversal in the posterior circulation territory.

Case Reports

Encounter 1
A 78-year-old right-handed white male with a history of paroxysmal atrial fibrillation developed sudden-onset right-side weakness and difficulty speaking. He arrived at a local emergency room 30 minutes after symptom onset. A noncontrast head computed tomography showed a cord-like hyperdensity in the left middle cerebral artery (MCA) trunk. International Normalized Ratio (INR) was 1.1. Intravenous tissue plasminogen activator (tPA) administration was considered. However, during the placement of a Foley catheter, the patient sustained urethral injury resulting in massive hematuria. As a result, intravenous tPA was felt to be contraindicated.

The patient was then transferred to our medical center for a higher level of management, arriving 3 hours after symptom onset. Blood pressure (BP) was 144/76 mm Hg, and heart rate (HR) was 80 to 110 with an irregularly irregular rhythm. The National Institutes of Health Stroke Scale (NIHSS) score was 18, comprising drowsiness, a left gaze preference, right homonymous hemianopsia, global aphasia, and right hemiplegia including facial weakness. The MRI done according to a protocol described previously was performed 4 hours after symptom onset (Figure 1) and showed an area of high signal intensity in the left basal ganglia region on DWI. The time to peak of the residue function (Tmax) perfusion images revealed greater distribution of high signal intensity area than the one of DWI, indicating extensive hypoperfusion with a diffusion–perfusion mismatch. A decision was then made to attempt recanalization of the occluded artery with intra-arterial thrombolysis. Complete recanalization of the left M1 was noted on postprocedure angiography 45 minutes after initiation of the procedure (6 hours and 35 minutes after symptom onset).
onset). The patient received tPA for a total dose of 11 mg. NIHSS score immediately after the procedure was 6, comprising slight drowsiness, mild right-sided hemiparesis, and dysarthria. The postprocedure MRI, including gradient echo sequences, was done 5 hours after recanalization was noted and revealed petechial hemorrhage in the left basal ganglia area. Clinically, the patient demonstrated progressive improvement in neurological outcome. On discharge home from the rehabilitation unit, 2 weeks after the event, his activities of daily living were independent and NIHSS was 2.

**Encounter 2**

Five months later, the same patient noted sudden onset of vertigo and generalized weakness and then slumped to the floor. He arrived at his local emergency room 1 hour after symptom onset. Because of his decreased level of consciousness, he was intubated for airway protection. INR was 1.2. The patient’s wife reported that he had been taken off his warfarin 5 days before admission in preparation for a hernia repair procedure, which was performed 2 days before the index event. The patient was again transferred to our medical center for further care, and arrived ≈2 hours after symptom onset. BP was 158/88 mm Hg and HR 100, again with an irregularly irregular rhythm. The initial neurological examination showed that the patient was comatose with occasional decerebrate posturing and had no purposeful movement of any of his extremities. The NIHSS score was 26. The patient was not treated with intravenous tPA because of his history of a recent stroke with hemorrhagic transformation. An MRI performed 3 hours after symptom onset, according to the same protocol used during the first encounter, revealed areas of acute ischemia in the bilateral pontine basis, right greater than the left, as well as in the cerebellar vermis. Hypoperfusion areas were detected in the entire brain stem and cerebellar hemispheres, particularly the pons and cerebellar vermis (Figure 2). Magnetic resonance angiography displayed occlusion in the proximal basilar artery. Because the patient was within a 6-hour therapeutic window but had a recent stroke with hemorrhagic transformation, thereby making the option of intra-arterial thrombolytic therapy administration also risky, it was decided to attempt vessel recanalization using a mechanical clot retrieval device. On left vertebral angiography, complete occlusion of the proximal basilar artery was noted. The device was then deployed with successful retrieval of the clot from the occluded vessel at 5 hours and 40 minutes after symptom onset. Clinically, the patient became more responsive, with occasional purposeful movements of all 4 extremities. Day 7 MRI showed reversal of much of the ischemic areas. The DWI lesion volume in the brain stem and cerebellar vermis had decreased from 6.9 cc at baseline to 3.1 cc at day 7. The areas of hypoperfusion in the pons and cerebellar vermis had completely resolved (Figure 3). At the time of discharge to an acute rehabilitation unit 16 days later, neurologic examination revealed a right sixth nerve palsy, dysarthria, and mild left-sided weakness. NIHSS

Figure 1. DWI shows an increased signal intensity focus in the left basal ganglia region. The Tmax perfusion images demonstrate a greater area of “at-risk” hypoperfused tissue, with substantial diffusion-perfusion mismatch in the left MCA territory.

Figure 2. At the time of the second event, with acute basilar artery occlusion, the preprocedure DWI reveals foci of acute ischemia in the pons, right more than the left, and cerebellar vermis. The perfusion images demonstrate low perfusion areas in the entire pons, the cerebellar vermis, and cerebellar hemispheres.

Figure 3. The day 7 postprocedure DWI shows almost complete resolution of the ischemic areas, although some scattered high-intensity signal foci remains in the pons and cerebellar vermis. No evidence of hypoperfusion is noted on the perfusion images.
was 7. He was able to ambulate short distances with the aid of a walker.

**Discussion**

This case is unique for 2 reasons. First, for this unfortunate patient, lightning struck twice. On 2 consecutive occasions, potentially devastating ischemic strokes occurred, and in spite of an early arrival to a hospital emergency room, he was deemed ineligible for the only Food and Drug Administration–approved therapy for ischemic stroke because of concerns about its safe administration in the setting of hemorrhagic risk. As a result, 2 distinct neurointerventional therapies were successfully used on both occasions.

The second distinctive aspect of this case was that the high-intensity signal area noted on baseline DWI in the brainstem and cerebellar vermis was almost completely resolved on the day 7 postprocedure DWI. Although documented DWI reversal has been reported in the anterior circulation and multimodal MRI has been demonstrated to be of great help in identifying pretreatment diffusion–perfusion mismatch in the posterior fossa, this is the first reported case, as far as we know, of reversal of a diffusion abnormality in the posterior circulation territory. This finding reinforces the concept that early recanalization can salvage not only regions of the ischemic penumbra but also areas of initial ischemic injury itself and extends it to the posterior fossa as well.

Acute basilar artery occlusion, as occurred in our case, can result in substantial morbidity and mortality and can be a clinical challenge. Larger series of patients with diffusion MRI reversal in the posterior circulation will be required to determine how frequently this phenomenon occurs and what its predictors might be. For the category of patients who present within the 3-hour intravenous tPA window but who have contraindications to its safe administration, emerging endovascular therapies and emerging physiologically based neuroimaging modalities may provide health personnel with an opportunity to reduce the devastating consequences of ischemic stroke and perhaps the potential to even reverse its initial effects.

**References**


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