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## Reversal of a 30-h fixed deficit with carotid angioplasty and stenting: technical note

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**Abstract** We describe the case of an 82-year-old man with a fixed neurological deficit of 30 h duration. A left hemispheric perfusion deficit was found on perfusion/diffusion imaging studies in conjunction with an ipsilateral carotid stenosis documented by cerebral angiography. Carotid angioplasty with stent placement was performed and resulted in dramatic clinical improvement. Carotid stenosis can cause acute hemodynamic hypoperfusion with a symptomatic reversible clinical deficit.

### Introduction

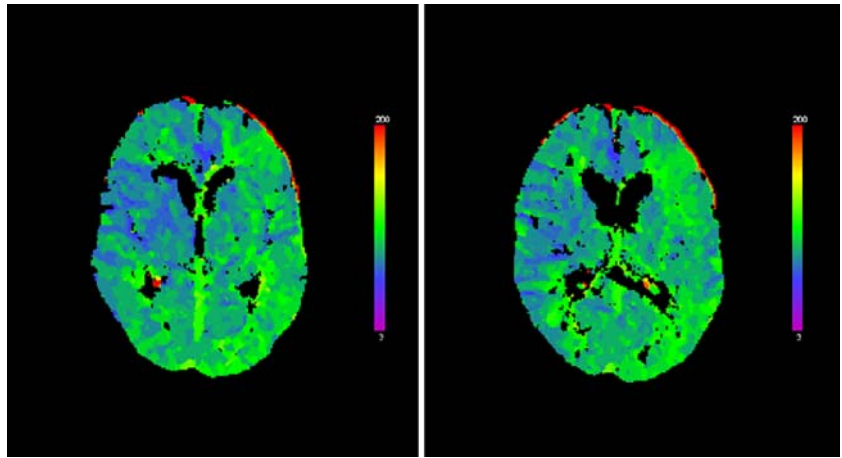
Patients with an acute neurological deficit lasting for longer than 6 h are not considered candidates for stroke therapy [1]. Nevertheless, an aggressive investigation may be worthwhile to identify patients who might benefit from revascularization. Computed tomographic (CT) perfusion and magnetic resonance (MR) diffusion/perfusion imaging techniques that demonstrate the volume of noninfarcted brain tissue at risk—the so-called ischemic penumbra—and the volume of tissue already infarcted provide real-time imaging data on which interventional decisions can be based. Patients with small areas of completed infarction and large areas at risk may remain candidates for interventional therapy. In this technical note, we illustrate the utility of an aggressive imaging investigation in the evaluation of a patient with a 30-h fixed neurological

deficit. We describe the potential of carotid stenosis to cause hemodynamic compromise of an isolated hemisphere and the application of endoluminal revascularization to reverse this deficit.

### Case description

An 82-year-old white man with a history of hypertension presented to our institution with a 4-day history of intermittent episodes of right-sided weakness and speech difficulties. Eighteen hours before his admission, the patient had developed a fixed right hemiplegia and mixed aphasia; on admission, his National Institutes of Health Stroke Scale (NIHSS) score was 18. A noncontrast CT scan of the brain demonstrated no hemorrhage or infarction. CT perfusion imaging revealed an increased time-to-peak

**Fig. 1** CT perfusion imaging reveals left hemispheric increased time-to-peak on two consecutive axial acquisitions

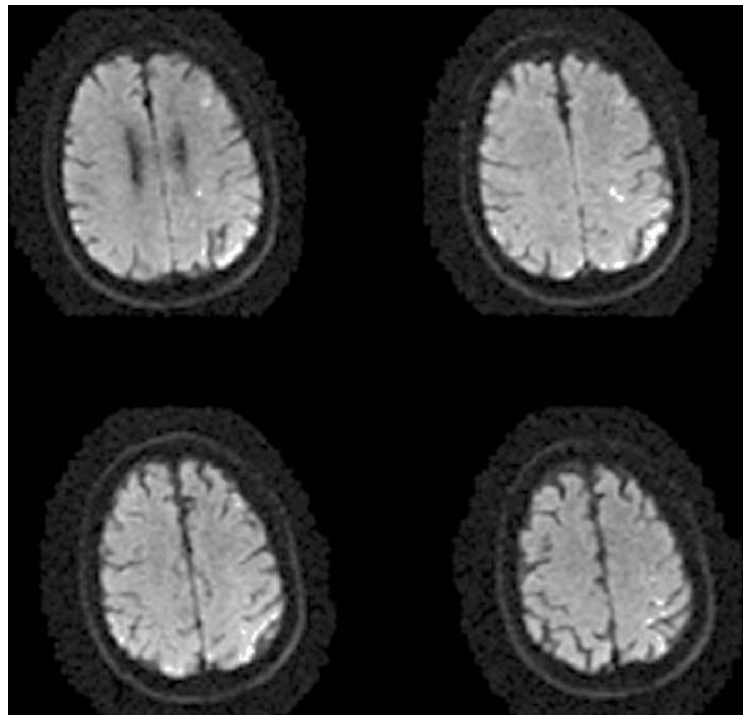


(delayed or reduced flow) in the entire left hemisphere; there was no black hole (which indicated the presence of persistent flow) (Fig. 1). Diffusion-weighted MR imaging demonstrated scattered left hemispheric hyperintensities (Fig. 2). A cerebral angiogram revealed approximately 75% stenosis of the left internal carotid artery; no other abnormalities of the intracranial vessels were documented during this study (Fig. 3). The left internal carotid artery supplied the left middle cerebral artery. No anterior cerebral artery filling was noticed from that side. Although the patient had a fixed neurological deficit persisting for 30 h, we elected to perform carotid angioplasty with stent placement to treat the left carotid artery stenosis because the functional imaging sequences had demonstrated significant left hemispheric hypoperfusion without a large infarction. Typically,

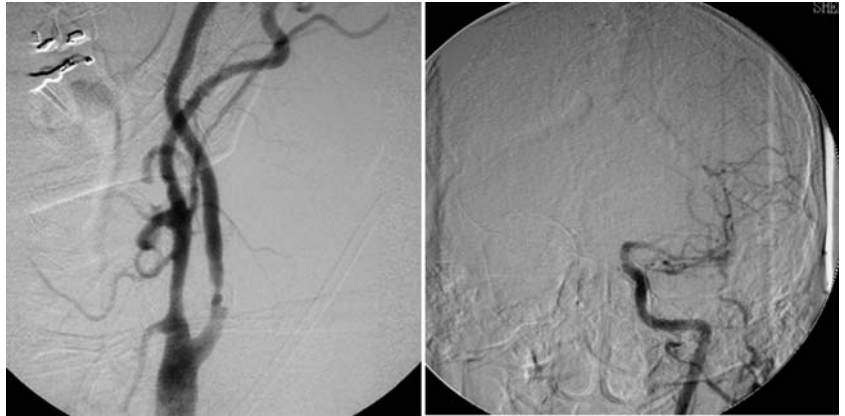
the presence of a large black hole on a perfusion study precludes us from undertaking aggressive therapy since this corresponds to infarcted tissue and, in our experience, carries a high risk of hemorrhage transformation.

After the placement of a 6F sheath in the right common femoral artery, a bolus dose of heparin (50 U/kg) was administered intravenously; an activated coagulation time above 250 s was obtained. A 6F guide sheath was placed into the left common carotid artery and used as the stenting platform. A 190 cm filter wire (EPI, Boston Scientific, Natick, Mass.) was used to cross the lesion and the filter was deployed successfully in the distal left internal carotid artery. Pre-stent deployment angioplasty was performed with a 3.5×30 mm balloon (Crossail, Guidant, Indianapolis, Ind.). Stenting was performed using a 10×37 mm stent

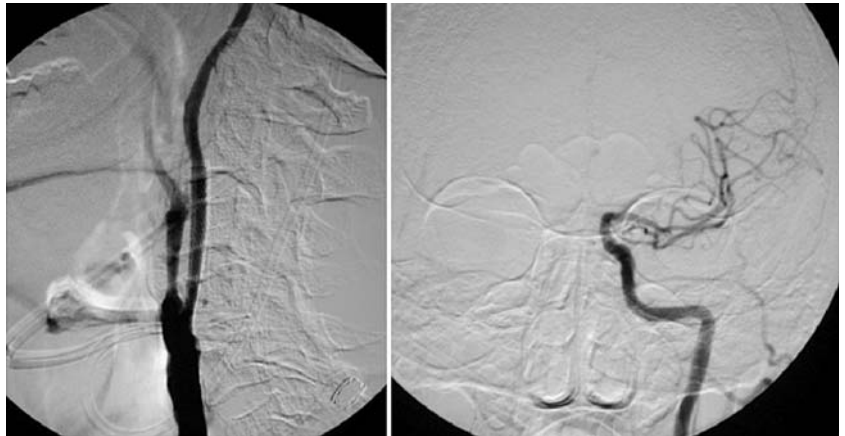
**Fig. 2** Diffusion-weighted MR images (axial acquisitions) reveal multiple, pinpoint left frontal hyperintensities



**Fig. 3** Cerebral angiogram shows severe left internal carotid artery stenosis and normal intracranial vessels (*left lateral view, right anteroposterior view*)



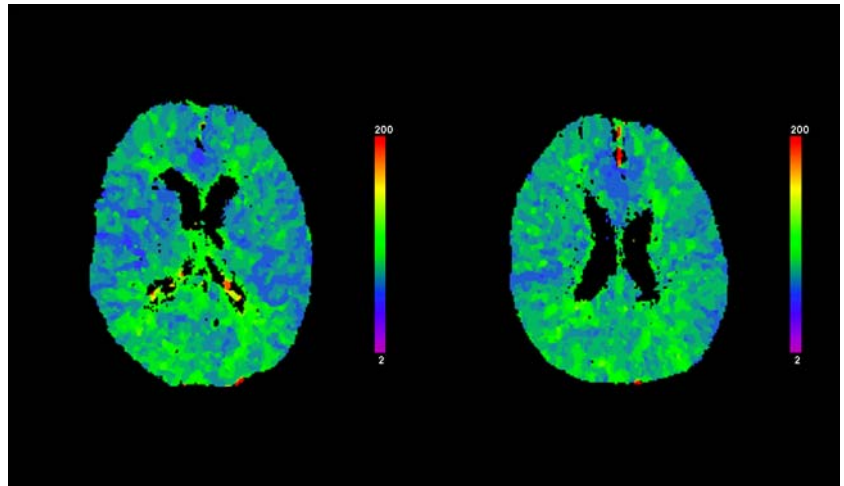
**Fig. 4** Cerebral angiogram after left carotid angioplasty with stent placement shows good revascularization and stent coverage of the stenotic lesion (*left lateral view, right anteroposterior view*)



(WallStent, Boston Scientific), which was deployed to cover the extent of the lesion. Poststenting dilation was performed using a 4×30 mm balloon (Aviator, Cordis, Miami Lakes, Fla.). After dilation, the filter was retrieved, final cervical and intracranial angiographic images were obtained (Fig. 4) and the procedure was terminated. The patient tolerated the procedure well.

Postprocedure CT-perfusion imaging documented normalization of the time-to-peak values in the left hemisphere (Fig. 5). The patient made a dramatic recovery following the procedure. He was discharged on the third day after stenting with an NIHSS score of 1 (secondary to a subtle right pronator drift) and was able to resume normal activities.

**Fig. 5** CT perfusion study obtained 48 h postprocedure shows normalization of time-to-peak (compared with the preprocedure images) on two consecutive axial acquisitions



## Discussion

The ideal imaging modality for acute stroke would accurately represent the volumes of both infarcted brain tissue and noninfarcted brain tissue still at risk. Given the brief time interval for successful treatment, the imaging technique must be performed quickly and easily. On routine CT scans, findings of cerebral ischemia, if present, are subtle in the acute setting [2]. However, MR imaging has proven to be extremely sensitive to the volume of cerebral ischemia and the findings have been correlated with the clinical severity [3]. Higher accuracy and better sensitivity of diffusion-weighted MR imaging (compared with CT scanning) for identifying acute infarction have been reported [4]. Diffusion-weighted imaging, which involves the detection of restricted interstitial water diffusion related to cellular energetic failure, membrane dysfunction and subsequent cytotoxic edema within the ischemic territory [5], coupled with apparent diffusion-coefficient imaging, better demonstrates irreversibly injured ischemic tissue [6]. Abnormalities detected on diffusion-weighted imaging are unlikely to regress and usually correspond with the infarction volume, although reversible changes have infrequently been reported [7].

At our institution and other stroke centers, CT perfusion has emerged as a rapid and accurate imaging modality in the setting of acute stroke. By acquiring sequential CT images of the brain during the intravenous administration of a bolus of contrast material, CT perfusion can be used for accurate assessment of cerebral infarction and penumbral tissue [8]. The ability to measure these perfusion parameters has been made possible with the development of high-speed helical or spiral CT scanners and software improvement. Data are acquired by measurement of the integrated change in tissue density following the intravascular injection of a contrast agent to calculate cerebral blood volume. Perfusion parameters can be quantified to characterize various degrees of perfusion in penumbral tissue. Some limitations of the CT perfusion technique are

inferior image quality in patients with low cardiac output and poor assessment of the posterior fossa and brainstem. Renal failure represents a contraindication because of the amount of contrast material needed for CT perfusion imaging. However, even with these limitations, CT perfusion has greatly influenced the management of anterior circulation ischemic stroke at our institution. Patients who show decreased time-to-peak but no black hole in a given anatomic territory are considered candidates for intraarterial treatment, even outside the traditional 3 to 6-h window.

The case presented here represents a unique situation in which a proximal carotid artery stenosis in the context of an isolated middle cerebral artery with the absence of anterior communicating artery and other collateral pathways resulted in hemodynamic compromise. The patient's fixed neurological deficit was reversed by reperfusion of the left middle cerebral artery territory through carotid angioplasty and stent placement.

CT and MR perfusion imaging studies delineate the penumbra of salvageable parenchyma in cerebral infarction. Using these studies, it may be possible to identify patients who might benefit from aggressive revascularization after the traditional therapeutic window has closed. Interestingly, in this case, carotid stenosis was found to have hemodynamic repercussions with secondary clinical symptoms that resolved after treatment.

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