Serial FLAIR Imaging After Gd-DTPA Contrast Pitfalls in Stroke Trial Magnetic Resonance Imaging

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Background—Most MRI protocols for stroke trials comprise 2 successive fluid-attenuated inversion-recovery (FLAIR) imaging acquisitions in which the first scan is done pre–Gd-DTPA contrast while the second is within the contrast clearance window.

Summary of Report—A 68-year-old male was diagnosed as having hyperacute right middle cerebral artery infarct and a subacute chronic small left occipital cortical infarct. The latter turned from hypointense to strikingly hyperintense on the second FLAIR image, resembling the picture of an acute-on-chronic infarction or hemorrhage. However, the second DWI and CT refuted either of these.

Conclusions—Image contrast using FLAIR in acute stroke trial imaging may also be affected by T1 effects of Gd-DTPA in chronic infarcts. (Stroke. 2003;34:797-798.)

Key Words: contrast media ■ magnetic resonance imaging ■ stroke, acute ■ thrombolytic therapy

MRI applied during stroke trials for neuroprotective drugs often requires a baseline scan and a repeated scan within 4 to 8 hours after intravenous thrombolytic/placebo administration.1 The second scan may be open to misinterpretation if it is not kept in mind that this scan, unlike the first, collects post–contrast Gd-DTPA effects because of remnant (incompletely cleared) Gd-DTPA still in the circulation from the perfusion series in the first scan. We illustrate this with the following case for the fluid-attenuated inversion-recovery (FLAIR) sequence.

Case Report
A 68-year-old male trial volunteer was clinically diagnosed with acute ischemic stroke and referred for MR imaging. Informed consent in accordance with the local ethics committee was obtained from the patient. The baseline scan and a follow-up scan 5 hours later both comprised the following 4 sequences: diffusion-weighted imaging, FLAIR imaging, 3-dimensional time-of-flight MR angiography, and dynamic perfusion-weighted imaging (bolus administration of 20 mL Gd-DTPA contrast agent). The patient’s serum creatinine was normal; thus, renal function was presumably normal (ie, no kidney problems).

Besides a hyperacute right middle cerebral artery (MCA) infarct, the first MRI scan at 4 hours after ictus revealed a subacute chronic small left occipital cortical infarct, which was clinically silent to the patient. This had mild FLAIR hyperintensity (the Figure, a) and was hypointense on the diffusion-weighted trace image (not shown). In addition, an unenhanced CT scan (Figure, b) performed earlier at 2 hours after ictus at the Accident and Emergency Department also showed an obvious hypointensity not expected for an acute infarct. Interestingly, this old left occipital infarct became strikingly hyperintense (as did the acute right MCA infarct) on the second MRI scan at 9 hours after ictus on the FLAIR sequence (Figure, c). Close scrutiny revealed increased signal in the subarachnoid spaces of the superior cerebellar sulci and preptontine cistern as well. However, the left occipital area remained hypointense and unchanged on the diffusion-weighted trace image (Figure, d), ruling out the differential diagnosis of an interval acute-on-chronic infarction or hemorrhage from reperfusion. The left occipital infarct remained unchanged on a second unenhanced CT scan (not shown) at 26 hours after ictus.

Discussion
In infarctions, a nonintact blood-brain barrier2 allows paramagnetic contrast agents to diffuse into the extracellular space and to alter T1 relaxation locally. The FLAIR image of the nonacute occipital infarct in the Figure, a became conspicuously bright in the Figure, c on the repeated scan. This raised clinically relevant questions of whether there was interim acute-on-subacute/chronic infarction or hemorrhage into a nonacute infarct after intravenous thrombolytic/placebo. These differentials were refuted by a subsequent CT scan showing no change in appearance of the left occipital infarct. Hence, T1 changes due to incomplete clearance of the contrast agent were felt to be the cause. The pharmacokinetic information of Gd-DTPA in the systemic circulation is such that 83% of the dose is eliminated renally at 6 hours after injection.3 The amount remaining (not eliminated)
causes the signal change. How do T1 changes affect the FLAIR image contrast? The FLAIR sequence produces heavily T2-weighted images with cerebrospinal fluid suppression by using the inversion recovery scheme and acquiring the image data after a time delay (called the inversion time), when the longitudinal magnetization of the signal from cerebrospinal fluid is 0. Any other tissue with T1 similar to that of CSF then also appears strongly attenuated. This is true for the nonacute occipital lesion in the Figure, a. On the other hand, the Gd-chelate still remaining in the lesion may change T1 of the water molecules significantly. If standard inversion times are used, this results in incomplete suppression in the FLAIR image and hence in a hyperintensity, which may confuse the unwary (Figure, c and d). Similarly, the interval increase in conspicuity of the acute infarct on a serial (postcontrast) FLAIR sequence obtained in stroke trial MR imaging may be a result of T1 contrast effects in addition to pathological evolution of hyperacute to acute ischemia/infarction or hemorrhagic conversion.

References
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Stroke. 2003;34:797-798; originally published online February 6, 2003; doi: 10.1161/01.STR.0000056528.05390.58

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/34/3/797

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