

Transient Ischemic Attacks: Added Specificity from Modern MR Imaging

Jeffrey L. Sunshine

AJNR Am J Neuroradiol 2002, 23 (1) 4-5 http://www.ajnr.org/content/23/1/4

This information is current as of June 21, 2025.

4 EDITORIALS AJNR: 23, January 2002

occupying an immediately vital and strategic position. Our proficiency in integrating imaging techniques that illustrate both the morphologic and physiologic characteristics of the brain is paramount and is a currently evolving strategy for daily clinical practice at some academic institutions. This combination of information gained from diffusion tensor imaging and several MR imaging techniques that depict the physiologic behavior of the brain, including functional MR

imaging, spectroscopy, and cerebral vascular imaging, promotes the exciting prospect of understanding functional and morphologic network aberrations in the clinical presentation of many neurologic disorders.

LEIGHTON P. MARK, MD JOHN L. ULMER, MD Medical College of Wisconsin Milwaukee, WI

Transient Ischemic Attacks: Added Specificity from Modern MR Imaging

In this issue of the *AJNR*, Rovira and colleagues (page 77) present diffusion-weighted MR findings from a series of patients after transient ischemic attacks (TIAs). Data were collected on lesions detected with diffusion-weighted MR imaging within 10 days of symptom onset in a group of 58 patients. In this study, TIA was defined as stroke symptoms that completely resolved within 24 hours. Most patients manifested lesions, either in deep parenchymal foci or involving the cortex, despite the symptom resolution. The percentage of that majority exceeds previously defined percentages in other reports.

A TIA has generally referred to a patient with clinical stroke that then resolves within the first day after onset. Colloquially this has varied greatly. For some, the time period for recovery shortens, even down to a 1-hour increment for return to a normal baseline of neurologic function. Of greater concern have been variations of definition regarding the symptoms, where for some any deficit that resolves may suffice, but for others only specific dysfunction registers. Then of course we are all faced with great variation among specialists who evaluate not only the initial set of symptoms but also the effectiveness of the recovery. Surely the neurologic examination by a neurologist will differ from that of a primary care provider or a busy emergency room doctor.

Investigation of patients with TIA with diffusionweighted MR imaging has varied widely in the timing of image acquisition relative to the onset of symptoms. Even in Rovira et al's study, the imaging occurred any time within the first 10 days after the neurologic event. Indeed, only a small minority of patients underwent imaging examination in the first 2 days after onset. In a previous report, the imaging occurred in the first 3 days after TIA (1). Range of timing limits the study of the precise cause of infarct and does not yet answer to the sensitivity of the technique in the first hours after onset when therapeutic interventions are under consideration. Sensitivity at early onset remains a concern, as data from our series of MR imaging in hyperacute stroke patients and from others investigators show us that a substantially low number of patients imaged very

acutely will have false-negative diffusion-weighted imaging findings (2).

The duration of the transient symptoms in patients with TIA seems to correlate directly with the likelihood of positive findings on diffusion-weighted images. The current study reports average symptom duration of 1 hour for those patients without any imaging evidence of ischemia compared with 7 hours for those with a lesion on diffusion-weighted images. This reinforces the intuitive conclusion that longer standing deficits, even when they resolve, do represent more substantial insults. The current investigation should also pique our interest, because many of the lesions identified had cortical involvement. Thus, we should not consider a TIA as limited to deep perforating artery territories.

The TIA remains an important and timely health issue for us all, as it represents a leading diagnosis for emergency room visits and admissions to hospitals. Of greater concern, patients evaluated for TIA often "bounce back" with either repeat transient events, delayed permanent cerebral infarcts, or even fatalities (3). Therefore, TIA represents a prevalent, high-risk diagnosis for our patient population. However, TIA remains a diagnosis based on symptoms and their resolution, and until recently, lacking in objective findings so useful for classification, characterization, and comprehension of the underlying disease processes.

The advance of diffusion-weighted MR imaging has provided exactly this objectivity, and not surprisingly we have encountered a wide range of variations beneath the catchall term of TIA. The imaging data add needed specificity to the evaluation, for now we can distinguish those who have undergone a true transient phenomena, even when lacking image-based sequelae, from those who have had infarction, although the natural redundant neurologic pathways have quickly compensated for the loss. We must continue to pursue the application of advanced imaging of patients with TIA, perhaps for example, with evaluation of the perfusion parameters in affected regions. We must strive to define those who have ischemia that may produce infarct, and further define those lesions that will produce permanent debilitation

in order to justify intervention to attempt repair. Our imperative should remain to better understand the pathologic behavior of TIAs, and importantly, to more strongly lead our clinical colleagues to the best care that can be offered to our collective patients.

JEFFREY L. SUNSHINE, MD University Hospitals Cleveland and Case Western Reserve University Cleveland, OH

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Diffusion-Weighted Imaging in the Distinction of Benign from Metastatic Vertebral Compression Fractures: Is This a Numbers Game?

As radiologists, one of the frequent problematic diagnostic dilemmas we face is trying to distinguish a benign from malignant fracture. Often I find myself feeling embarrassed when reflecting upon my response to a clinician when posed with the question, "Is this vertebral fracture benign or malignant?" On the surface, it seems not only a reasonable question, but with recent advances in MR imaging, a question one might assume to be answered quite easily. Unfortunately, to date, MR imaging has not increased our specificity to a point that a confident answer can be given on a routine basis.

Vertebral metastasis is common and can be seen in 10% of all patients with cancer. Benign osteoporotic compression fractures are also common, particularly in the elderly and in those patients receiving longterm steroid therapy. MR imaging is excellent in the assessment of the bone marrow. This is particularly true in images obtained in adults in which the high T1 signal intensity of normal fatty marrow provides an inherent contrast that allows for the easy depiction of marrow-replacement processes. Typically, malignant marrow lesions that have increased water will appear hypointense on T1-weighted images and hyperintense on conventional spin-echo images or fat-suppressed T2-weighted images such as short-TI inversion recovery and chemically fat-suppressed T2-weighted images. The basic problem, however, is that acute and subacute benign fracture with marrow edema results in a similar alteration of the marrow signal intensity. Therefore, the presence of signal intensity changes in the marrow on conventional images is not helpful in distinguishing acute to subacute fractures from malignant fractures.

Structural changes of the vertebral bodies and changes surrounding the vertebra have also been used to distinguish benign from malignant fractures. Adjuvant MR findings have been used to suggest that a malignant fracture may be present, such as complete replacement of the vertebral body marrow, involvement of the posterior elements, and an associated epidural or paraspinal mass. Unfortunately, these findings are not specific and can be seen with benign fractures. Conversely, lack of these associated find-

ings does not exclude the presence of a malignant fracture. Benignity can be suggested if the vertebral signal abnormality only consists of a linear signal abnormality confined to the vertebral endplate. Contrast-enhanced MR imaging has not helped, because both benign and malignant vertebral fractures may enhance. The enhancement encountered in a benign fracture is presumably related to leakage of contrast agent through compromised vessels and the presence of granulation tissue.

In 1998, Baur et al (1) took a unique approach to this problem, studying 30 patients with 39 vertebral compression fractures. They used a time-reversed fast imaging sequence based on steady-state free precession (SSFP) to produce diffusion-weighted images. This SSFP sequence overcame many of the problems that prevent the performance of satisfactory echoplanar-based diffusion-weighted images in the spine but used a relatively low b value (165/mm²) to achieve an adequate signal-to-noise ratio. In their study, all benign vertebral fractures were hypo- to isointense to adjacent normal bone marrow, and all malignancyrelated fractures were hyperintense to normal bone marrow on diffusion-sensitized images. The explanation of their findings was as impressive as their results. Baur et al tested the hypothesis that the free mobility of water was different between acute benign fractures and pathologic fractures. The reduced mobility of water in pathologic fractures was the result of tumor cell accumulation and a subsequent reduction in the interstitial space. Increased mobility of water, on the other hand, was attributed to an increase in the interstitial space in relation to edema or hemorrhage in benign fractures. Although the results were promising, Baur et al indicated that bone marrow diffusion needed further investigation in a larger series of patients and that further study was necessary to clarify the changes of diffusion in the subacute and healing phase of fractures.

In an accompanying editorial, LeBihan (2) pointed out that further confirmation of these findings was necessary with proper quantitative analysis, which could not be performed with the SSFP technique employed by Baur et al. The basic problem was that