Letters to the Editor

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Gadolinium-Enhanced Magnetic Resonance Imaging in Multiple Infarction

To the Editor:

The interesting report of Miyashita et al.1 deserves two comments. In that study, nine patients with recent lacunar strokes, who were noted to have multiple small infarcts on computed tomography (CT) and magnetic resonance imaging (MRI), underwent enhancement studies with iodinated contrast medium and gadolinium-diethylenetriaminepentaacetic acid (Gd-DTPA), respectively. A comparison showed that recent lacunes were better visualized in Gd-DTPA-enhanced MRI than with contrast CT, and the authors recommended a larger study to confirm the results.

First, there is evidence that patients with acute strokes have poorer outcomes if given iodinated contrast medium for CT,2 particularly in those with enhanced ischemic lesions. A likely explanation is that the abnormal permeability of the blood–brain barrier in ischemic lesions allows the contrast medium to reach the cerebral parenchyma (reflected by enhancement) and to exert a neurotoxic effect. Under such circumstances, one would hardly imagine that approval from an ethics committee for a larger study of a similar nature could be obtained.

Second, the cost-effectiveness of identifying a recent lacune is questionable. In most cases of stroke, the site of the lesion can be accurately diagnosed clinically, and the purpose of nonenhanced CT or MRI is to ascertain whether the lesion is an infarct or hemorrhage and to exclude other conditions such as subdural hematoma or tumor. To identify a recent lacune in the presence of multiple small infarcts, as suggested by the authors, will more than double the cost of an expensive imaging investigation, yet add little to its management. Hence, the use of Gd-DTPA enhancement in MRI, while of academic interest, will probably find very limited application.

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References


The following is in reply:

To the Editor:

We appreciate Dr. Yu’s comments. Gadolinium diethylenetriaminepentaacetic acid (Gd-DTPA)–enhanced magnetic resonance imaging (MRI) is not a routine examination and may be indicated only for patients whose clinical conditions cannot be clarified by computed tomography (CT) or nonenhanced MRI alone. The application of Gd-DTPA–enhanced MRI in stroke patients is, therefore, limited as a matter of course. In our stroke care unit, more than 700 patients with acute stroke were admitted during the last 3 years, and only 45 of them, the majority of whom had multiple lacunar infarctions, received Gd-DTPA–enhanced MRI.

While the requirement of Gd-DTPA–enhanced MRI was not frequent among our patients, its use did provide helpful information and contributed to better management of these cases. Patients with multiple lacunes usually have residual neurologic deficits attributable to old lesions and may show sudden deterioration of symptoms due to hypotension or dehydration rather than to recurrent lacunar infarction. Such cases may be the best indication for Gd-DTPA–enhanced MRI. Although the expense of Gd-DTPA–enhanced MRI is twice that of CT or nonenhanced MRI alone, cost-effectiveness may be of little importance if the examination is clinically useful.

As confirmed in several studies,1,2 toxicity of Gd-DTPA is slight. Furthermore, the amount of Gd-DTPA required for enhancement is almost one-tenth that required for iodinated contrast media.3 Thus, Gd-DTPA–enhanced MRI may be considered to be much safer than contrast CT.

None of 45 patients receiving Gd-DTPA–enhanced MRI in our stroke care unit showed deterioration of symptoms or other adverse effects. However, since the penetration of any foreign substance through the blood–brain barrier may further increase ischemic tissue damage, unnecessary contrast studies should always be avoided. Dr. Yu seems to be misinterpreting our report2 because careful review of our paper shows that we are not recommending a large group of enhancement studies at all.

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References


Platelet Levels of Glutamate and Aspartate in Normal Subjects

To the Editor:

The excitatory amino acids glutamate and aspartate are believed to mediate neuronal cell damage in certain neurologic diseases including cerebral ischemia. Platelets are thought to be a useful
model for the study of neurotransmitter metabolism. Therefore, we attempted to measure the concentration of glutamate and aspartate in platelets. We studied 12 healthy subjects ranging in age from 20 to 47 years (six men, six women). All were free of drugs known to affect platelet function for at least 2 weeks before the study. Blood (8 ml), drawn from an antecubital vein from subjects resting in the supine position, was placed into plastic tubes containing citrate. Platelet-rich plasma (PRP) was obtained after centrifugation at 165g for 15 minutes. Platelets were counted using a Baker-810 platelet analyzer. Thereafter, 1 ml PRP was centrifuged at 2500g for 15 minutes to obtain a platelet pellet, which was resuspended in 1 ml saline and sonicated for 5 minutes; 30 mg/ml salsalate acid was added to deproteinize the platelet preparation. This preparation was then centrifuged at 1200g for 5 minutes, and an aliquot of the supernatant was injected into a Millipore/Waters high-performance liquid chromatography column (Milford, Massachusetts) connected with a Perkin-Elmer fluorescence spectrometer (Norwalk, Connecticut). Glutamate and aspartate were measured using norvaline as an internal standard.

The mean±SD platelet glutamate and aspartate levels were readily detectable at 0.30±0.15 and 0.47±0.14 μmol/10^10 platelets, respectively. Accordingly, we suggest that platelets can be used as a more available substrate to study the function of these excitatory amino acids in neurologic disease including stroke. Furthermore, since glutamate and aspartate have toxic properties and have been implicated in the pathogenesis of ischemic cell damage, we should perhaps consider platelets as an alternate source of these amino acids in ischemic stroke.

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Clinical Disagreement on the Diagnosis of Transient Ischemic Attack: Is the Patient or the Doctor to Blame?

To the Editor:

Like many other clinical diagnoses, that of transient ischemic attack (TIA) is subject to considerable interobserver disagreement. One reason for these variations may be that different clinicians obtain different information from the patient; another is that the observers may interpret the same history differently. Physicians participating in interobserver studies tend to stress the former and overlook the latter. In a previous study, 72 patients with possible TIAs were separately interviewed by two neurologists, randomly paired from a group of 18. We found that of the eight cases in which the observers disagreed on the diagnosis, six disagreements could be attributed to differences in interpretations, whereas only two resulted from a difference in acquired information.

To further elucidate whether the doctor or the patient is the major source of clinical disagreement, we also performed the following experiment. Two simulated patients were added to the study population of 72 patients, without the participating neurologists' knowledge. These two simulated patients were actresses who were thoroughly trained in giving consistent information under all circumstances. One of them, aged 53 years, was taught a history of a single attack of clumsiness of one arm and disturbed articulation, which had come on suddenly and had lasted 10 minutes. The other, 58 years old, was supposed to have experienced two kinds of attacks. First, she had noticed a rather vague visual disturbance of her left eye, "like looking through a steamy pane," and lasting for 1 minute. Some weeks later she had experienced a tingling sensation in her right arm, spreading in a few minutes to her face and leg; this attack lasted 30 minutes. Neither attack was followed by headache, but the second simulated patient had had migraine as a young adult.

Each simulated patient was interviewed by four pairs of neurologists. One of the 16 observers appeared slightly suspicious after having interviewed a simulated patient, but the other neurologists noticed nothing unusual. The observers were asked to adhere to recommended rules for the diagnosis of TIA, which are based on internationally accepted criteria and have been used for many years in our department. These criteria were included as a supplement to a checklist on which the symptoms were to be recorded in detail. According to the recommended criteria, the attack of the first simulated patient qualified as a TIA, whereas the two attacks of the second simulated patient did not. All eight pairs of neurologists showed complete uniformity in the description of the nature and time course of the individual symptoms. Yet in the first simulated patient, seven observers concluded "TIA," while one concluded "no TIA." In the second simulated patient, six observers concluded "no TIA," whereas two observers from different pairs concluded "TIA." Altogether, only five of the eight pairs agreed on the diagnosis (agreement corrected for chance: χ²=0.25 compared with χ²=0.77 for the real patients).

The results from our small experiment confirm that differences in the interpretation of symptoms are probably more important as a source of disagreement than differences in the content of the history. This implies that the consistency of the diagnosis of TIA could be improved if the diagnostic guidelines are thoroughly discussed and consistently followed. The patient is not always to blame.

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References
1. Sisk C, Ziegler DK, Zilenti T: Discrepancies in recorded results from duplicate neurological history and examination
Platelet levels of glutamate and aspartate in normal subjects.
G D’Andrea, A R Canazi, F Ferro-Milone, R Joseph, S Grunfeld and K M Welch

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