Acute stroke

Magnetic resonance perfusion diffusion mismatch, thrombolysis, and clinical outcome in acute stroke

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Use of PWI–DWI mismatch as an inclusion criterion for thrombolytic treatment in acute stroke

In this issue, Kane *et al*¹ (see p 485) report a systematic study of the value of magnetic resonance perfusion-weighted imaging (PWI)–diffusion-weighted imaging (DWI) mismatch to predict the clinical response to thrombolytic treatment in patients with acute stroke. The author's main conclusion on this crucial question is straightforward: it is not prime time to use the identification of mismatch as a guide to therapeutic decisions. Buttressing this recommendation on methodology used by the Cochrane Study Group, the authors correctly ward off the premature acceptance of inadequately proven techniques. This systematic analysis is timely and concise but there are reasons to forecast a wider use of MRI-guided stroke treatment in the near future.²

A primary goal of multiparametric MRI in acute stroke is to identify signals consistent with viable brain tissue. PWI–DWI mismatch has been proposed as a reliable marker of tissue viability that could be used in selected patients to extend the current thrombolytic window of 3 h.³ In the accompanying article, the detection of mismatch had low sensitivity and specificity to predict outcome. Moreover, the presence of mismatch was associated with a twofold increase in the odds of infarction growth, although expansion of the infarction was also seen

in patients without mismatch. Unfortunately, the study could not unravel the mechanisms that led to infarction growth in relation to the variable presence and extent of mismatch, use of thrombolytics, rate of recanalisation or some combination of these factors. Regardless of the administration of thrombolytics, the presence of mismatch was a harbinger of bigger lesions at follow-up, but a too small sample size resulted in wide CIs. Surprisingly, infarction growth on MRI was not associated with worse functional outcome, unlike studies that used CT scanning for outcome prediction.⁴ Although this could in part be explained by a type II error, another explanation could be that the outcome measures selected in the studies of the systematic analysis (Modified Rankin Scale (MRS) and Barthel Index (BI)) had low sensitivity to clinical changes. Recent observations suggest that the volume of mismatch, DWI, PWI or T2-weighted abnormalities on MRI, is better correlated to neurological impairment (ie, National Institutes of Health Stroke Scale score) than to measures of activities of daily living (MRS, BI).⁵ The latter outcome measures may be more susceptible to the topography of the infarction, not only its volume, and the confounding effects of comorbidity.

The recently reported Diffusion and Perfusion Imaging Evaluation for

Understanding Stroke Evolution study provides the stronger indication that for patients with stroke treated 3–6 h after onset with thrombolytics, baseline MRI findings (mismatch) can identify subgroups that are likely to benefit from reperfusion and can potentially identify subgroups that are unlikely to benefit or can be harmed.² The study was specifically designed to deal with the value of mismatch to predict outcome and its findings are therefore very relevant. Nevertheless, it is desirable that ongoing studies confirm these encouraging findings and refine current imaging protocols before planning to use mismatch as an inclusion criterion for thrombolytic treatment in acute stroke, as judiciously pointed out by Kane *et al*¹.

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