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Diffusion- and perfusion-weighted MRI in a patient with a prolonged reversible ischaemic neurological deficit

Received: 26 July 1999
Accepted: 20 September 1999

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Abstract We report acute and follow-up diffusion- and perfusion-weighted MRI (DWI, PWI) findings in a patient with a prolonged reversible ischaemic neurological deficit. PWI 12 h after the patient was last seen to be without symptoms revealed a large perfusion deficit in the left posterior MCA territory with a relatively inconspicuous and much smaller abnormality on DWI. Follow-up showed resolution of abnormalities on both DWI and PWI, and conventional MRI was normal, apart from a very slight abnormality, visible only on FLAIR images, at the centre of the initially DWI-positive region. These findings demonstrate the utility of PWI when used in combination with DWI to investigate the pathophysiology of transient ischemic syndromes.

Key words Magnetic resonance imaging, diffusion-weighted · Magnetic resonance imaging, perfusion · Stroke, acute

Introduction

Although there is a rapidly growing body of data on diffusion- and perfusion-weighted MRI (DWI, PWI) in acute stroke [1, 2], relatively little information is available on findings in transient ischaemic syndromes, in particular concerning PWI. In the largest study reported to date, Kidwell et al. [3] analysed DWI findings in patients with transient ischaemic attacks (TIAs). More than half the patients in this study had normal DWI and,

in those with positive DWI studies the lesions were small and relatively subtle. This suggests that DWI alone may not be sufficient to identify the pathophysiology underlying transient ischaemic syndromes.

We report a patient with a reversible ischaemic neurological deficit lasting over 24 h (prolonged reversible ischaemic neurological deficit; PRIND), with extensive abnormalities on PWI and only relatively minor changes on DWI, who made a full clinical and nearly complete tissue recovery on follow-up MRI.

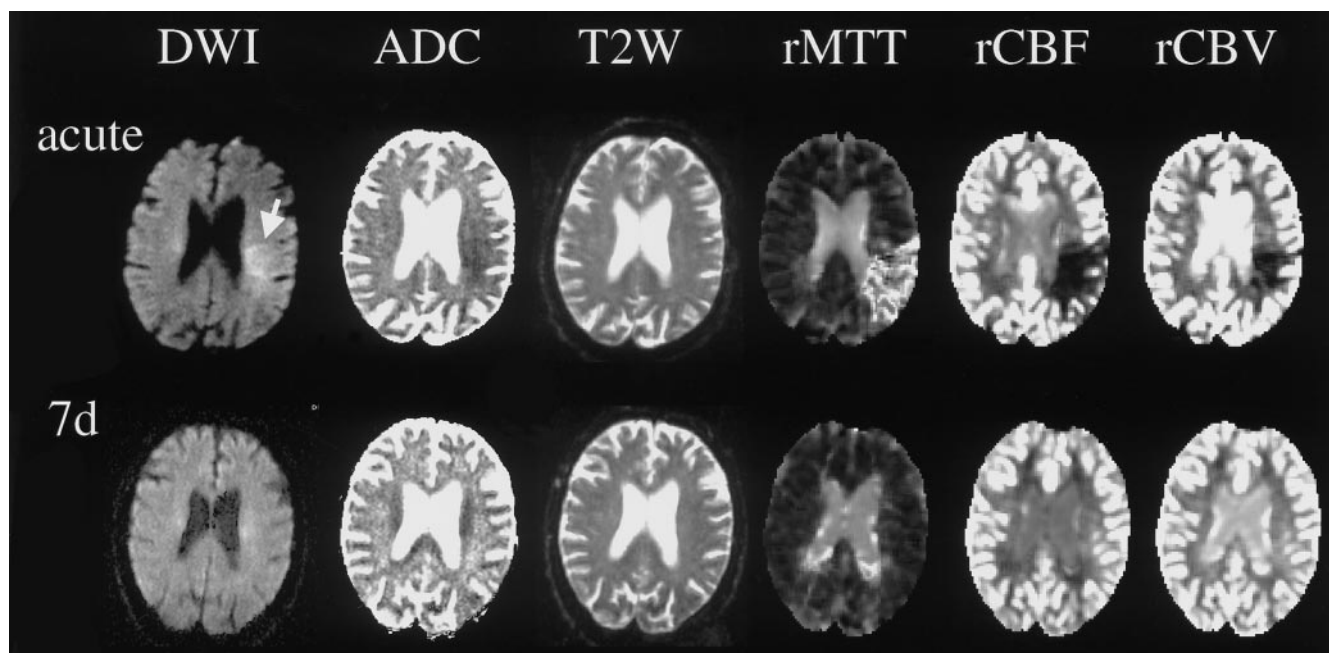


Fig. 1 In the acute phase (*top*), diffusion-weighted imaging (DWI) revealed a relatively inconspicuous lesion (*arrow*) with a decrease in apparent diffusion coefficient, but no infarct on T2-weighted images (T2W). A perfusion deficit was observed on all acute perfusion-weighted imaging maps: *MTT* mean transit time, *CBF* cerebral blood flow, *CBV* cerebral blood volume. The perfusion deficit was substantially larger than the abnormality on DWI, in particular on the MTT and CBF maps. Follow-up at 1 week (*7d, bottom*) showed complete resolution of the abnormalities on DWI and PWI

Case report

A 71-year-old man with a history of hypertension and mild hypercholesterolaemia was admitted 2 h after waking up with severe, predominantly sensory aphasia and mild right hemiparesis. The patient had last been seen to be without symptoms 12 h before presentation. His European Stroke Scale (ESS) score was 73/100.

MRI was obtained 30 min after his arrival at the emergency department, using a 1.5-T system. The protocol included fast spin-echo T2-weighted images, a single-shot echo-planar (EPI) diffusion-weighted sequence (DWI) and perfusion-weighted imaging (PWI). DWI was performed using TE (echo time) 100 ms; b_0 and 1000 s/mm²; field of view (FOV) 240 mm; matrix 96 × 128; slice thickness 5 mm, gap 1.5 mm; 20 slices. PWI was obtained using a gradient-echo EPI technique (TE 54 ms; FOV 240 mm; matrix 128 × 128) with 40 consecutive measurements in each of 12 slices at intervals of 2 s during injection of 0.2 mmol/kg gadolinium-DTPA. PW images were processed to generate maps of relative mean transit time (rMTT), relative cerebral blood flow (rCBF) and relative cerebral blood volume (rCBV) after deconvolving the tissue concentration-time curve with an arterial input function, obtained by manually choosing pixels over the middle cerebral artery (MCA) of the unaffected hemisphere [4].

PWI revealed a large perfusion deficit in the left posterior MCA territory, in which abnormal regions could be identified on

the rMTT, rCBF, and rCBV maps (Fig. 1). The volume of the abnormal regions, and the difference in signal intensity between the two sides were calculated for both affected cortical regions and white matter (Table 1). The abnormal region was slightly larger on the MTT maps than on the rCBF maps and substantially larger than on the rCBV maps. The perfusion deficit was clearly less severe in the cortex than in the underlying white matter region.

DWI showed a relatively inconspicuous diffuse increase in signal, confined to white matter, within the perfusion deficit. The apparent diffusion coefficient (ADC) in the abnormal area was decreased to 60% of that of the corresponding contralateral area. The T2-weighted images were normal.

Investigation revealed a patent foramen ovale, with right-to-left shunting, shown by both transesophageal echocardiography and transcranial ultrasound, indicating that a paradoxical embolism may have underlain the cerebral ischaemic event. There was no evidence of current deep venous thrombosis. Other cardiac investigations and extracranial Doppler ultrasound yielded normal results.

Since the patient was not eligible for i. v. thrombolysis (due to the unknown time of onset) he received only standard therapy (i. v. heparin, blood pressure management) and no neuroprotective agents or other investigational drugs. Several hours after the initial MRI study he started to improve clinically. The next morning only minor amnesic aphasia was still detectable and 4 days later he had returned to normal.

The follow-up MRI studies obtained at 7 days (T2-weighted images, DWI and PWI) and 3 months (T2-weighted spin-echo and FLAIR images, DWI) showed resolution of the DWI and PWI abnormalities and no residual lesion was seen on fast spin-echo T2-weighted images. However, there was slightly increased signal on the FLAIR images at the centre of the initially DWI-positive region (Fig. 2).

Table 1 Analysis of abnormalities on MRI

	Diffusion-weighted imaging	Regional mean transit time	Regional cerebral blood flow	Regional cerebral blood volume
Lesion volume (ml) ^a	6.3	76.2	51.3	12.7
Signal intensity ratio between cerebral hemispheres ^b				
Cortex	0.97	2.37	0.45	0.97
White matter	0.56	1.76	0.27	0.53

^a Determined by manually tracing the edge of the regions and multiplying the calculated area by the slice thickness plus interslice gap

^b Ischaemic region/corresponding region of unaffected hemisphere

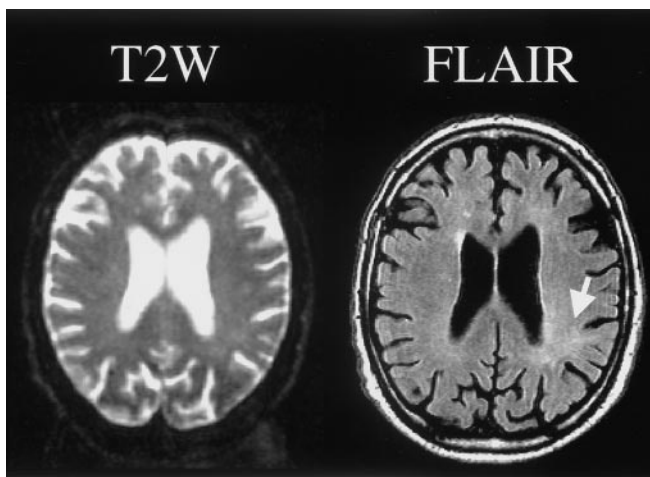


Fig. 2 At 30 days FLAIR images revealed a slight increase in signal in the area which appeared abnormal on acute DWI, but no frank infarct. No infarct was found on T2-weighted images

Discussion

We demonstrate that PWI can be useful in addition to DWI for the assessment of the pathophysiology underlying reversible ischaemic syndromes. The acute perfusion abnormality in our patients was far more extensive than the relatively subtle DWI lesion. In fact, the latter – although unequivocal – was appreciated only with hindsight, and might have been overlooked without knowledge of the PWI results.

Previous studies using either conventional MRI or CT in patients with clinically reversible ischaemic syndromes have shown that 2–48% have relevant infarcts at follow-up, despite clinical recovery [3, 5]. It has been suggested that these conditions may be more appropriately classified as “cerebral infarcts with transient symptoms (CITS)” [6]. In the remainder of patients both conventional MRI and CT remain normal.

DWI is known to be much more sensitive to ischaemic tissue injury, but the percentage of patients with a TIA and abnormal findings on DWI within 3 days of the onset of symptoms has also been reported to be less

than 50%, and in some of those with abnormal DWI no relevant infarct was found at follow-up [3]. Our case suggests that combined PWI and DWI may be considerably more sensitive than DWI alone in transient ischaemic syndromes. There are theoretical considerations supporting this view. Abnormalities on DWI are generally thought to be a marker of severe ischaemic tissue injury, and in practice these regions usually evolve into infarcts [7–9]. PWI, on the other hand, has been shown to be sensitive enough to detect less severely hypoperfused regions, which may have impaired function, contributing to the neurological deficit, but are still viable [10]. It is therefore reasonable to assume that most patients with truly reversible ischaemic damage will show substantially more extensive abnormalities on PWI than on DWI (or no abnormality at all on DWI), while they are clinically affected.

The acute DWI and PWI findings in our patient did not allow us to predict the benign clinical course with full recovery. Even patients with acute abnormalities on PWI, but not on DWI may develop relevant infarcts and permanent clinical deficits [11]. However, both of these patterns (PWI deficit > DWI lesion or no DWI lesion) appear to be compatible with *potentially* (almost) complete tissue recovery, provided spontaneous or treatment-induced reperfusion occurs early enough.

Acknowledgement Dr. Neumann-Haefelin was sponsored by a grant from the DFG (Ne 569/2-1).

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