

DWI Identifies Thalamic and Hippocampal Involvement in Patients with Prolonged Ictal Activity

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INTRODUCTION

DWI has been shown to detect early changes related to ictal activity. Transient reductions of the apparent diffusion coefficient (ADC) have been detected with DWI in experimental focal ischemia with periinfarct depolarisations, as well as in models of spreading depression and epilepsy. Several case studies have demonstrated reduced ADCs in patients with focal status epilepticus[1,2]. Another recent report also claimed to have observed transient ADC changes immediately after single seizures[3]. We report the use of acute DWI in a series of 16 patients presenting with acute clinical deficits due to prolonged focal ictal activity.

METHODS

MRI was performed with one of two clinical 1.5T MRI systems (Siemens SONATA, Siemens VISION) according to the following protocol: 1. Transverse, coronal, and sagittal localizing sequences followed by transverse oblique contiguous images (slice thickness 5 mm) aligned with the inferior borders of the corpus callosum (sequences 2.-5.). 2. Proton density (PD)- and T2-weighted (TSE 2620 ms/14 ms/85 ms, FOV 180x240 mm², matrix size 192x256) 3. T1 - weighted SE (530 ms/ 12 ms, FOV 180x240 mm², matrix size 192x256) 4. DW EP-SE (TR 4000 ms/TE 110 ms, b =0/160/360/640/1000 s/mm², FOV 240 mm², matrix size 128x128, sequential application of 3 separate diffusion sensitizing gradients in perpendicular directions).

RESULTS

One patient had a previously diagnosed temporal lobe epilepsy with hippocampal sclerosis, while the remaining 15 patients had symptomatic epilepsy due to other primary acute or chronic cerebral pathologies. All patients were examined temporally close to prolonged ictal activity, mainly showing different degrees of alterations of consciousness or orientation. We identified different patterns of DWI changes: Acute DWI hyperintensity limited to the hippocampal formation was found in five patients; acute DWI hyperintensity limited to cortical areas adjacent to the pathology but clearly involving "healthy" tissue was found in two patients. In the other nine cases DWI alterations were either in the hippocampus and the thalamus (5/16), in the hippocampus, the thalamus and the cortex (1/16) or in the cortex and the thalamus (3/16). Figure 1 and 2 show the consistency of acute hippocampal and thalamic involvement on DWI images.

DISCUSSION and CONCLUSIONS

In all patients, prolonged reversible clinical ictal features and EEG findings were present correlating frequently exactly to the site of DWI abnormalities indicating that consequences of prolonged focal epileptic activity were detected by DWI. This close spatial correlation of initial DWI and EEG results suggest pathophysiological links between ictal brain activity and early parenchymal abnormalities. As demonstrated in experimental studies, metabolic compromise due to prolonged ictal overactivation may cause lactate accumulation and focally reduced water diffusion. Besides the clear involvement of hippocampal structures in epilepsy, several lines of evidence suggest that dorsal thalamic nuclei are also involved in various epilepsy syndromes. Focal increase of blood flow has been found in seven patients with absence epilepsy in a PET study[4]. Postictal T2-hyperintensity was noted in the dorsomedial thalamus on T2-weighted MRI in a patient with a right parietal seizure focus[5]. This DWI phenomenon provides further evidence for the immediate involvement of both, mesial temporal lobe and dorsomedial thalamic structures in epilepsy.

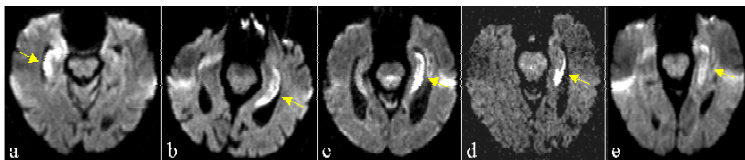


Figure 1: Diffusion-weighted MRI depicting involvement of the dorsal medial thalamic structures in patients 2, 4, 5, 13 and 14 (left to right) in almost identical areas across the five patients (yellow arrows). Extratemporal cortical hyperintensity in patients 2 and 5 is to be appreciated (green arrow).

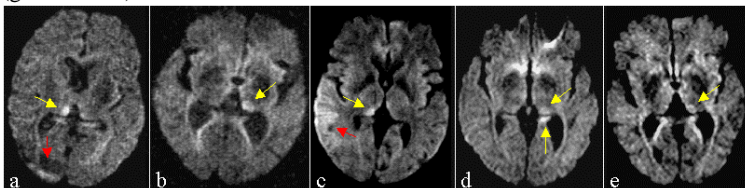


Figure 2: Diffusion-weighted MRI demonstrating involvement of the hippocampus in patients number 3, 4, 8, 13 and 14. High signal abnormality is noted in the right respectively, the left hippocampus (left to right, arrows). Diffusion-weighted MRI hyperintense image artefacts can occasionally be appreciated in the area of the temporal bone.

References

- [1] Lansberg MG, O'Brien MW, Norbash AM et al. MRI abnormalities associated with partial status epilepticus. *Neurology*. 1999; 52:1021-1027
- [2] Wieshmann UC, Symms MR, Shorvon SD. Diffusion changes in status epilepticus. *Lancet*. 1997; 350:493-494
- [3] Hufnagel A, Weber J, Marks S et al. Brain diffusion after single seizures. *Epilepsia*. 2003; 44:54-63
- [4] Prevett MC, Duncan JS et al. Demonstration of thalamic activation during typical absence seizures using H2(15)O and PET. *Neurology*. 1995; 45:1396-1402
- [5] Nagasaka T, Shindo K, Hiraide M et al. Ipsilateral thalamic MRI abnormality in an epilepsy patient. *Neurology*. 2002; 58:641-644