Diagnostic features of acute DWI abnormalities of different origin in the hippocampus

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Introduction

Diffusion-weighted MRI (DWI) and perfusion-weighted MRI (PWI) have in many places become standard diagnostic sequences that are mainly applied in acute stroke patients, but also in other situations of diagnostic uncertainty as they also provide information about acute tissue damage due to other cerebral pathologies. The hippocampus, as part of the limbic system is a particularly interesting anatomical structure not only because of the critical role in learning, memory and emotional behaviour but also because it is affected by various pathologies in acute and chronic neurological disorders. In particular, acute DWI and ADC (apparent diffusion coefficient) abnormalities in the hippocampus are a frequent finding in acute neurological syndromes. Analysis of the features of these hippocampal changes may give diagnostic clues to the underlying pathology, especially in neurological disorders other than ischemic stroke. We analysed DWI and PWI changes in patients with acute hippocampal syndromes.

Material and Methods

From our MRI database (1996 - 2006) with images of patients treated in our Neurology Department we analyzed those with acute DWI hyperintensity and corresponding ADC reduction in the hippocampus (n = 124). Seventy-two were hospitalized because of transient global amnesia (TGA), 15 patients were examined after prolonged epileptic seizures (mainly complex partial status epilepticus, CPSE), and 37 of these patients were diagnosed with acute stroke in the posterior cerebral artery (PCA) territory. In all patients MRI was performed on a 1.5 Tesla MR System (Vision or Sonata, Siemens Medical Systems, Germany) with echo planar (EP) hardware (gradient power 25 mT/m, rise time 83 mT/m/ms) using a standardized protocol including diffusion-weighted (b=1000), MR angiography and perfusionweighted (dynamic susceptibility) sequences.

Results

We identified 3 distinct hippocampal lesion patterns in the three patient groups: a) In 72 TGA patients we detected small, punctate DWI lesion in the lateral aspect of the hippocampal formation on either side or bilaterally. Lesions were rarely noted in the hyperacute phase, but all became visible at 24-48h. These lesions were not accompanied by alterations of the perfusion state or vessel pathologies. b) In patients with CPSE regional hyperintensity on DWI, and a reduction of the ADC with close spatial correlation of focal hyperperfusion on PWI was observed. Subtle but obvious signs of hyperperfusion on TTP, MTT and CBV maps were confirmed by additional SPECT studies in 3 patients. Similar signal changes were commonly found in the pulvinar of the ipsilateral thalamus. c) In the stroke subgroup we identified 4 patterns of acute ischemic lesions of the hippocampus involving either the complete hippocampus (commonly with additional large lesions affecting the complete PCA territory), only the lateral aspect of the hippocampus along the complete length of the hippocampal body and tail, the dorsal part of the hippocampal body, or the lateral border with circumscribed lesions. Interestingly, hippocampal stroke lesions never occurred as single acute ischemic lesions; in all patients additional DWI lesions outside the hippocampus (e.g. occipital lobe, internal capsule) were found. On MRA persistent PCA occlusion or stenosis was identified in 72.5% of patients with corresponding hypoperfusion on PWI (Figure 1).

Discussion and Conclusion

The hippocampus can be affected in patients with TGA, CPSE and stroke causing high signal on DWI with reduction of the ADC in each case. However, on the basis of the 3 distinct lesions patterns, MRI can also provide additional differential diagnostic information, especially in those cases, in which clinical symptoms alone do not enable definite diagnosis. While in stroke cytotoxic edema is believed to cause a decrease in ADC, the potential development of cytotoxic edema and signal changes in the other etiologies is less well understood. In epilepsy these changes are thought to be a consequence of compromised energy metabolism, as prolonged ictal activity is known to increase glucose utilization, the increase of which is not adequately matched by the enhanced blood flow. The underlying pathology of the changes in TGA remain unclear; while delayed ischemic or hypoxic mechanisms are assumed, it is still difficult to perceive these lesions as typical vascular ischemic changes. The different features of DWI abnormalities observed on patients with stroke, CPSE and TGA can provide diagnostic clues to the underlying pathology particularly when clinical information is scarce (Table 1).

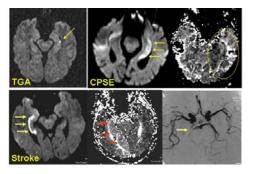


Figure 1: Examples of acute DWI signal changes in a patient with transient global amnesia (TGA; top left) with a circumscribed, dot-like lesion in the hippocampus detected 36 hours after clinical symptoms: MRA and PWI in this patient were normal. In a patient with disorientation, stereotypical speech patterns and EEG-proven complexpartial status epilepticus (CPSE; top right) DWI shows hyperintense signal in the left hippocampus accompanied by hyperperfusion on PWI. A patient with hippocampal stroke (bottom) shows hyperintense signal in the

Table 1: Common MRI and clinical constellations hippocampal DWI lesions

	TGA	lschemia	lctal
DWI	Circumscribed, occuring delayed, after 24-48 hours	Patterns according to vascular pathology	Typically involving lateral aspect of hippocampal body
MRA	Normal	PCA occlusion or stenosis	Strong PCA signal
PWI	Normal	Hypoperfusion	Hyperperfusion
Typical clinical features	Agitation; repeatedly asking questions concerning recent events	Typical symptoms of PCA stroke, mnestic features if left or both hippocampi involved	Prolonged disorientation and automatisms

hippocampus corresponding to the course of the longitudinal terminal segments of the hippocampal arteries. This patient had additional DWI lesions in the splenium, an occlusion of the PCA with hypoperfusion in the occipital lobe.