

Fast and robust lesion detection and assessment in acute ischaemic stroke patients from ADC and quantitative T2 mapping

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Introduction: Currently only 2 – 10% of acute stroke patients are thrombolysed, though 25% may be eligible for the treatment, with unknown onset time the key contra-indication. MRI holds considerable promise in pre-treatment classification. Recently ADC/T₂-FLAIR signal mismatch has been clinically trialled for patient stratification¹ but has relatively low sensitivity. Preclinical studies indicate that absolute relaxations times, T₁² and T₂, in combination with ADC make tissue status evaluation more robust and also provide an estimate of stroke onset time^{2,3}. Here we present a simple, rapid and automatic routine for the detection of ischaemic tissue from ADC mapping, and subsequent tissue characterisation by quantitative T₂ mapping in acute ischaemic stroke patients. We show that T₂ correlates differently with ADC between ischaemic and non-ischaemic tissue, and propose that this may be used as an additional restraint in distinguishing between differently affected regions of the ischaemic tissue, and different lesion types.

Methods: Patients were scanned 3-6 hours after stroke onset at 3 T with multi-echo GRASE (TE 20,40,60,80,100 ms) and 3-directional DWI ($b = 1000 \text{ mm}^2/\text{s}$). T₂ maps were computed by a voxel-wise mono-exponential fit, ADC maps calculated directly from the input images. ADC lesion detection was then applied by selecting voxels in a reciprocal ADC map with outlying high values. Clusters of voxels were accepted by the routine as a “lesion” only if that cluster had a volume larger than a user-defined threshold to exclude unreasonably small volumes unlikely to be an ischaemic tissue. The lesion(s) were then filtered according to their T₂ values, segmenting the ADC lesion into voxels with T₂ values within the range of normal T₂ values for that patient and those significantly higher. The overall correlations between T₂ (or another imaging parameter) with ADC were then calculated for the whole imaging volume, ADC lesion, and T₂-segmented ADC lesions. All parametric mapping, lesion detection and segmentation routines were written in-house using Matlab.

Results: Figure 1 shows an example of the automated lesion detection by ADC and segmentation by T₂. About half of the infarct volume has T₂ within a normal range (green), the other outlying high T₂ (red). A comparison of T₂ values in the ADC lesion to the rest of the imaging volume shows a clear increase in T₂, less apparent in other imaging metrics (Figure 2). We investigated the relationship between ADC and the T₂ by correlating their values after bin-ranging the ADC map to average over multiple observations in order that noise be removed. This showed that the usual positive ADC-T₂ correlation is altered in the lesion, with different behaviour in the T₂-normal and T₂-high regions. We propose that this is a proxy of tissue state in ischaemia and could be a characteristic feature to distinguish between different lesion types, such as reversible and irreversible ischaemia.

Conclusions: Stroke lesions can be rapidly and automatically detected in human patients, with quantitative T₂ mapping providing a simple and fast means of segmenting a lesion into regions of normal and non-normal relaxation behaviour. T₂-ADC correlations also show promise in distinguishing healthy from ischaemic tissue.

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References:

- 1 Thomalla *et al* Lancet Neurology 10; 978, 2011
- 2 Jokivarsi *et al* Stroke 41; 2335, 2010
3. Rogers *et al* NeuroReport 25; 1180, 2014

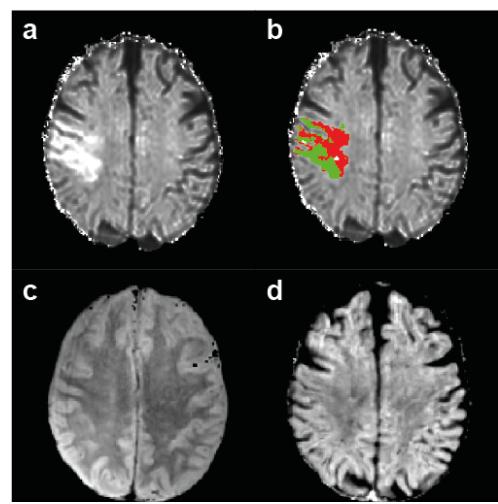


Figure 1 (a) Reciprocal ADC map, (b) reciprocal ADC map with automatically detected lesion after T₂ segmentation. Green = T₂ in normal range, red = T₂ high outlier. (c) I(0)Fitted image from T₂ fitting, (d) R₂ map

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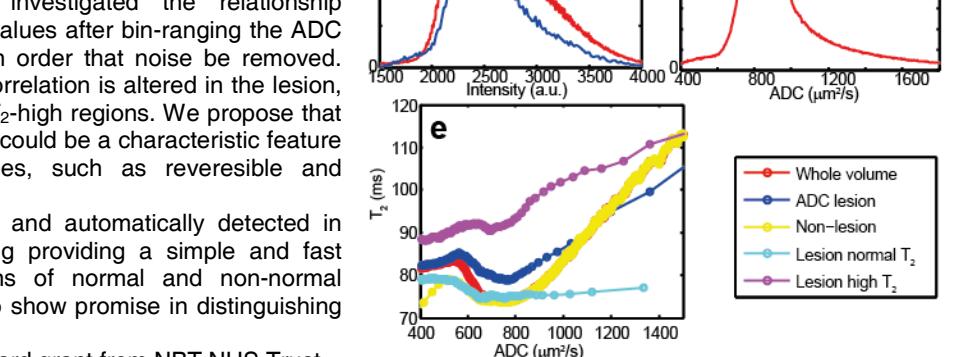


Figure 2. Distributions of parameters across the imaging volume and within the ADC lesion. (a) T₂, (b), I(0), (c) T₂-weighted intensity, (d) ADC (whole volume only). The y axis is a normalised frequency density in each case.