

Prediction of Tissue Recovery in Chronic Stroke Using Adaptive Models and Acute MR Information

Hassan Bagher-Ebadian^{1,2}, Marie Luby³, James R Ewing^{2,4}, Panayiotis Mitsias⁴, and Hamid Soltanian-Zadeh^{1,5}

¹Radiology, Henry Ford Hospital, Detroit, MI, United States, ²Physics, Oakland University, Rochester, MI, United States, ³National Institute of Neurological Disorders and Stroke, MD, United States, ⁴Neurology, Henry Ford Hospital, Detroit, MI, United States, ⁵CIPCE, ECE Dept., University of Tehran, Tehran, Iran

Target Audience: Neuroradiologists, neurologists, and biomedical scientists who are interested in studying stroke patients and therapeutic interventions of stroke.

Purpose: This pilot study demonstrates the application of adaptive models in predicting of tissue recovery in chronic stroke, using acute MR information.

Introduction: Magnetic resonance imaging (MRI) plays an important role in the diagnosis and management of acute ischemic stroke; it is sensitive and fairly specific in detecting the penumbra area that is used to predict stroke outcome [1]. Experimental and clinical studies indicate that the likelihood for progression to infarction in the penumbra of physiologically impaired, but potentially salvageable, tissue surrounding the central core of focal cerebral ischemia is an important factor in planning treatment and also evaluating treatment efficacy [1-2]. Thus, an analysis of multi-parametric MR images that detects and characterizes ischemic penumbra in the early stages of stroke may have profound clinical significance [1-2]. We have recently shown that the inelastic collision (IC) model [3], the Kohonen Multi-Parametric Self-Organizing-Map (KMP-SOM) [4], the Generalized Linear Model (GLM) [5] and an Artificial Neural Network [6], can all be adapted to information theory in order to construct a model-based algorithm for multi-parametric analysis. This pilot study demonstrates the application of these four adaptive models in constructing a probability map of tissue recovery in chronic stroke, using acute MR information.

Materials and Methods: Eleven treatment-naïve patients presenting with acute neurological deficit consistent with stroke, and with MRI studies within 24h of onset, were selected. The severity of the neurological deficit was assessed using the National Institutes of Health Stroke Scale (NIHSS) score at the time of each MRI study. MRI studies were performed at an acute time point (<24 h after stroke onset) and chronic time point (90 days after stroke). MRI studies were acquired on a 1.5 Tesla GE Signa MR scanner with echo-planar capability. Each MRI study consisted of axial multi-spin echo T2-weighted imaging (T2WI), T1-weighted imaging (T1WI), diffusion-weighted imaging (DWI), and Fluid Attenuation Inversion Recovery (FLAIR) with slice thickness of 6 mm and 7 mm. The field of view (FOV) was 240 × 240 mm. For T1 and T2 imaging, the matrix was 256 × 192 and for DWI 128 × 128. Additional parameters for each study were: (a) T1WI: TR/TE = 600/14 ms; (b) T2WI: TR/TE = 2,800/30, 60, 90, 120 ms; (c) axial DWI was performed using an echo-planar sequence, TR/TE = 10,000/101 ms, b-values = 1,000 s/mm², 1 NEX, FLAIR: TR/TE=9000/145. For MR Perfusion (MRP), 2D-EP/GR with 20 slices (6mm), 40 time points (2.2 sec), TR/TE =2200/45, 128 × 128. For each patient, two sets of co-registered images (T1, T2 –TE90, DWI and PD or FLAIR for non-MRP and Mean Transit Time, relative Cerebral Blood Volume, DWI and T2-TE90 for MRP) at the acute time point were put into the adaptive models (IC, KMP-SOM, ANN, GLM) to produce their corresponding multi-parametric maps. Hemodynamic maps (MTT and rCBV) were estimated from MRP data using Singular Value Decomposition in the required deconvolution. The difference between acute DWI and co-registered T2WI chronic lesions (initial lesion - chronic lesion at 3 months) was considered as the potential of lesion growth. The power of predicting the core and pattern of the T2WI chronic lesion in multi-parametric maps was used for evaluation and the robustness of the proposed adaptive models.

Results and Discussion: Figures 1 and 2 show that the patterns of the lesions in the probability maps for tissue recovery (2nd row in figure 1 and 2nd and 3rd rows in figure 2) estimated by the proposed adaptive models are well matched with their corresponding lesion patterns in the T2WI at chronic stage. The results of this pilot study confirm that, given acute MR information, adaptive models can reasonably estimate a probability map that estimates the long-term tissue growth. These results also imply that each method has a different prediction power that appears to be dependent on the condition of the tissue at risk as well as the systematic properties of the selected algorithm. Figure 2 presents the prediction power of ICM and KMP-SOM algorithms when these algorithms are presented with different combinations of MR modalities acquired from an untreated patient: DWI, T2WI, rCBV, and MTT, versus DWI, T2WI, T1WI, and FLAIR. The results of this study strongly support the idea of combining different MRI weightings to find the best combination of modalities for constructing a predictive model. We conclude that adaptive models are capable of identifying the ischemic growth, both in pattern and size, and may describe tissue viability. Since their predictions are strongly related to the clinical outcome, adaptive models can play an important role in the assessment of subacute therapeutic interventions, currently of great interest in the treatment of stroke.

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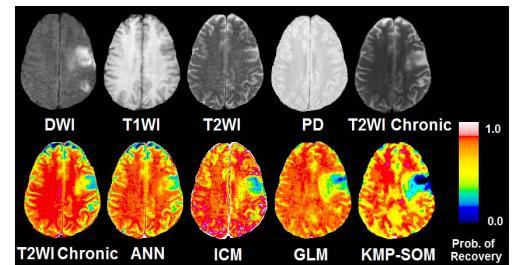


Figure 1: The acute DWI, T1WI, T2WI, PD and chronic T2WI of an untreated patient (first row) used for estimating the maps of probability of tissue recovery after 3 months (second row).

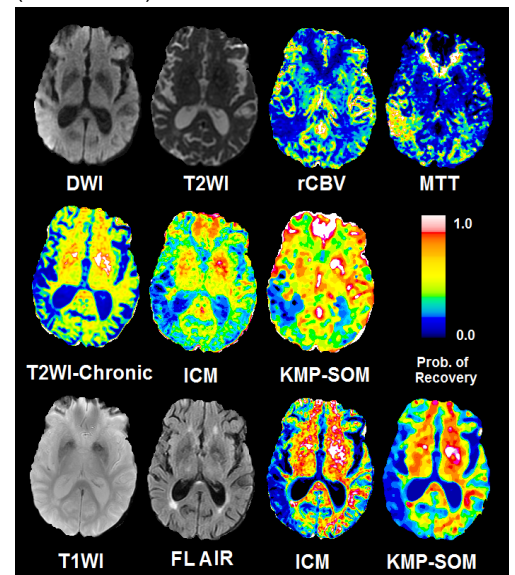


Figure 2: The last two images in the 2nd and 3rd rows represent the predicted maps of probability for tissue recovery after 3 months for an untreated patient using ICM and KMP-SOM techniques. These maps have been estimated from MRP and non-MRP modality combinations (shown in the 1st and 3rd rows)