

Predicting Final Infarct Size and Pattern of 3-month Lesion in MRI for Patients with Ischemic Stroke: Using Artificial Neural Network Technique

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Introduction

Early identification and discrimination of ischemic tissue status is essential for rational decision-making in acute brain stroke therapy [1]. In stroke the pattern of recovery in brain tissue and/or its likelihood for progression to infarction are considered to be the most important biomarkers for evaluating treatment efficacy [2]. Unsupervised clustering approaches *e.g.*, ISODATA (Iterative Self-Organizing Data Analysis Technique) can predict the eventual infarct size using acute information [3,4]. However, these methods identify major data clusters and assign individual pixels to one of these clusters. This approach, while useful, does not provide a pixel-by-pixel judgment as to the probability of eventual infarction; finding a point estimator for infarction remains an open problem [4, 5]. In this study, we hypothesized that, given a gold-standard map of T₂ in the chronic stage of stroke (3 months post-stroke), an Artificial Neural Network (ANN) might be trained to directly predict the size and pattern of the tissue fate similar to the T₂WI from the information available in acute MR images. An image set at the acute stage consisting of T₁ pre-contrast, T₁ post-contrast, T₂, Diffusion-Weighted and proton density was selected as the input to the ANN. The co-registered three-month T₂ map was considered to be the gold standard for the tissue fate. The training dataset consisted of 6 MRI sets from untreated stroke patients acquired at an average of 16:52 ±4 hours after onset. The ANN was trained, optimized and validated by the leave-one-out method. The results demonstrate that an ANN is a good candidate ($r=0.9670$, $p<0.0001$) for predicting the 3-month lesion pattern and size from the information in the acute MRI.

Materials and Method

Six patients presenting with acute neurological deficit consistent with stroke, with MRI studies within 24h of onset, were selected. Stroke onset was defined as the last time the patient was known to be without neurological deficit. 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 the acute time point (<24 h after stroke onset), and outcome time point (90 days after stroke). Patients were excluded if they had cerebral hemorrhage at the acute time point or a history of prior stroke. MRI studies were acquired on a 1.5-tesla GE Signa MR scanner with echo-planar capability (GE, Milwaukee, Wisc., USA). Each MRI study consisted of axial multi-spin echo T₂-weighted imaging (T₂WI), pre- and post-contrast (Gd-DTPA) T₁-weighted imaging (T₁WI) and diffusion-weighted imaging (DWI) with slice thickness of 6 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, 600, 300, 0 s/mm², 1 NEX. For each patient, five images (T₁ pre, T₁ post, T₂-TE90, DWI and PD) at the acute time point were selected to provide the essential input features to the ANN. All acute and chronic images were registered to T₂-TE90 as reference using Eigentool software [6]. To reduce mis-registration effects, all images were smoothed using a with a 5X5 low pass. To create features independent of the MR system gain, all images were normalized to their brain mean value. A feature set was generated and sampled from five acute images and put into a feed-forward multi-layer perceptron (MLP) with back-propagation training algorithm. The ANN was trained, optimized and validated by leave-one-out method. The AUROC value (Area Under Receiver Operator Characteristic curve) of the ANN was used to calculate the ANN performance and optimize the ANN structure.

Result

The optimal ANN ([5+1 bias];[10 +1bias],[1]) was found by maximization of AUROC (A_z=0.8152) for 1 hidden layer. As Figure 1 demonstrates, the ANN's predictions (2nd row images) are visually similar to their corresponding chronic T₂ images (1st row). More objectively, as the graph demonstrates, the predicted pattern and lesion size by the ANN are well correlated to their three-month lesion ($r=0.9670$, $p<0.0001$). Note that the continuity of the ANN output provides more information regarding the tissue viability compared to the clustering techniques such as ISODATA. Figure 2 presents the scatter plot of the ANN response *versus* T₂-weighted image at three months for 6 patients.

Discussion

A trained ANN capable of predicting the outcome, both in pattern and size, of a stroke from MR acute information can play an important role in tissue viability modeling. Since it is predictive of the outcome, such modeling may play an important role in the assessment of subacute therapeutic interventions, currently of great interest in the treatment of stroke. It may be possible to form a more clinically relevant prediction of outcome by including the NIHSS score as an input to the ANN, in addition to the stated feature set.

References

- [1] Lu M et al., J Magn Reson Imaging 2005;21(5): 495-502.
- [2] Shen Q et al., J Cereb Blood Flow Metab 2004; 24: 887-897.
- [3] Shen Q et al., J Cereb Blood Flow Metab 2005; 25:1336-1345.
- [4] Mitsias PD et al., American journal of Neuroradiology, 2004;(25):1499-1508.
- [5] Mitsias PD et al., Magnetic Resonance Imaging. 28th International Stroke Conference 2003;20-22.
- [6] Windham JP et al., J. Computer Assist. Tomography 1988; (12):1-9.

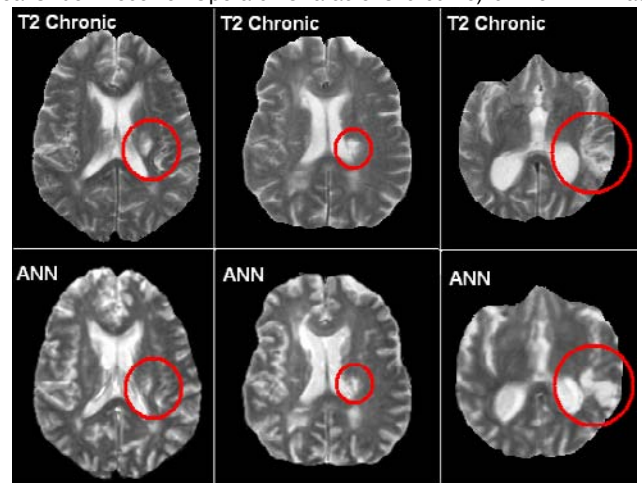
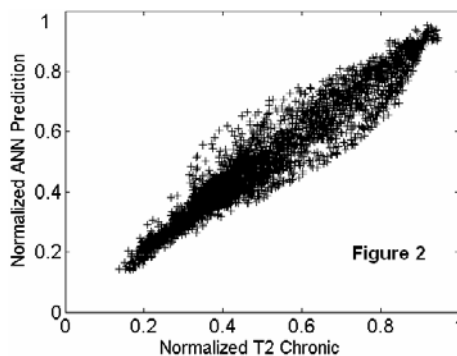


Figure 1