Automated Detection of Focal Cortical Dysplasia based on Textural, Statistical and Morphological Analysis of MRI

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Synopsis

An automated classifier to identify focal cortical dysplasia in patients with epilepsy was developed. The classifier was trained on 3D maps of first-order statistical and morphological models based on MRI characteristics of focal cortical dysplasia and 3D second-order maps constructed from second order texture analysis. Lesions were found in 15/18 patients. No lesional areas were identified in controls.

Introduction

Focal cortical dysplasia (FCD), a malformation of cortical development, is a frequent cause of medically intractable epilepsy. FCD lesions are characterized on T1weighted MRI by cortical thickening, blurring of the gray matter (GM)/white matter (WM) interface, and hyperintense lesion signal with respect to the rest of the cortex. In previous work, we introduced ¹and refined ² a technique for improving visual detection of FCD lesions by using first-order statistical and morphological models of their MRI characteristics. In the current study, we aimed to reduce the subjectivity associated with visual lesion detection by developing a computer-based classifier to perform automated lesion detection. The classifier was trained on the models used in our previous work, which incorporate visually discernable information, and results from second-order texture analysis, which can quantify patterns in the spatial distribution of gray level intensity that may be difficult to observe visually ³.

Methods

We studied 18 patients with FCD and medically intractable epilepsy, and 13 healthy controls. T1-weighted images were acquired on a 1.5 T Gyroscan using a T1-fast field echo sequence (TR=18, TE=10, 1 acquisition average pulse sequence, flip angle= 30° , matrix size=256x256, FOV=256, thickness=1 mm, ~170 slices, isotropic voxel size of 1 mm³). All patients had surgery and FCD was subsequently proven based on histological examination of the resected tissue. FCD lesions were manually segmented by an expert observer.

Three-dimensional maps of the first-order statistical and morphological models used in our prior study were constructed for each subject. These consisted of a cortical thickness metric based on the solution of Laplace's equation ⁴, gradient magnitude calculated over a moving gaussian kernel to model blurring of the GM/WM interface, and a "relative intensity" operator to model hyperintense T1 signal by measuring the gray-level intensity of each voxel relative to the intensity threshold between GM and WM (determined through histogram analysis of the entire volume). Three-dimensional texture maps were generated for each subject by constructing a gray-level co-occurrence matrix (GLCM) over a small, moving 3D region of interest (ROI) centered at each voxel within the MRI volume. A set of eight standard second order texture feature operators were then run on the GLCM from each ROI to generate a set of 3D maps for each subject.

To determine target classes for training purposes, T1 volumes were automatically segmented into CSF, GM, WM, GM/WM interface, and GM/CSF interface. For patients, the manual lesion labels were incorporated into the segmented volume. A Bayesian classifier was trained on the maps of the first-order statistical and morphological models and three second order texture features (angular second momentum, contrast, difference entropy, which were selected using Fisher's discriminant ratio) to classify voxels within a T1 volume as CSF, GM, WM, GM/WM interface, GM/CSF interface, or lesional. A leave-one-out approach was used whereby voxels for each subject were classified based on the 3D maps of the remaining N -1 subjects. The results of the classifier were compared to standard visual evaluation of pre-surgical MRI.

Results

The classifier correctly identified lesions in 15/18 patients, compared to 11/18 identified by standard pre-surgical evaluation (p<0.03). The figure presents a typical result from the classifier. No lesional areas were identified by the classifier in the control subjects. In 5 patients, the classifier identified additional lesional areas in the cortex that did not co-localize with the manual lesion labels. Retrospective visual review of conventional MRI for these cases did not reveal lesional tissue.



Conclusions

The classifier improves upon our previous work by providing an automated approach to lesion detection. A strength of the classifier is its consideration of first- and second-order information from the T1-weighted MRI volume. The finding that no lesional voxels were identified in any control subject is especially relevant in light of the fact that in some patients the classifier identified lesional clusters that did not co-localize with manual lesion labels. These clusters may indicate abnormal regions that are otherwise undetectable via conventional MRI analysis.

References

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