Automated detection of subtle focal cortical dysplasia using multi-contrast MRI

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PURPOSE

Focal cortical dysplasia (FCD) is a highly epileptogenic developmental malformation. Its surgical removal is the only effective treatment to control seizures, limits adverse effects on cognition, and reduces risks of injury and death. On MRI, FCD lesions present with cortical thickening and blurred cortico-subcortical interface; similar to histology [1], abnormalities may range from visible to the naked eye to more subtle ones. Despite advances in MRI analytics, current algorithms are not optimized to accurately detect subtle FCD lesions, a scenario in ~50% of referrals for pre-surgical evaluation [2]. The current work proposes a novel in vivo surface-based automated detection algorithm. Our method exploits the complementary diagnostic power of T1-weighted and T2-weighted FLAIR contrasts [3], together with a synthetic FLAIR/T1 ratio map designed to increase the sensitivity for co-occuring FLAIR hyperintensity and T1w hypointensity at the grey and white matter interface.

METHOD

Figure 1 summarizes our method.

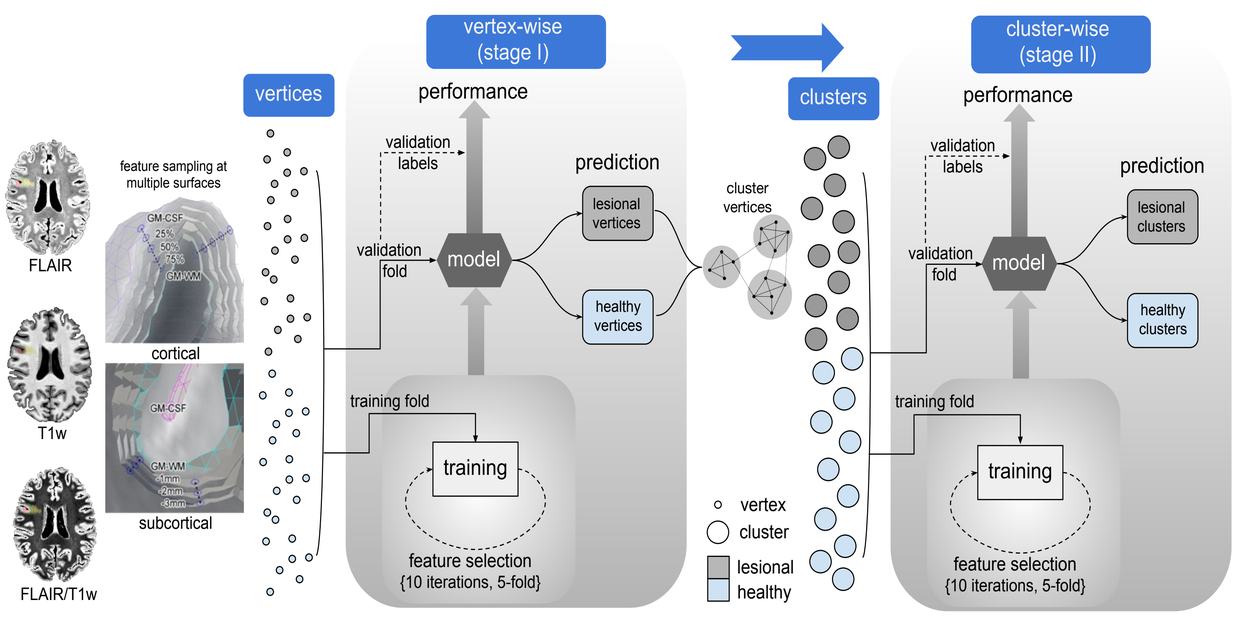

Figure 1. Vertex-wise and cluster-wise classification schema.

Based on 3T T1-weighted MRI (3D MPRAGE; voxel size: 1 x 1 x 1 mm^3) and 3D FLAIR (0.9 x 0.9 x 0.9 mm^3), we extracted cortical surfaces using an automatic algorithm [4] and measured cortical thickness, sulcal depth, curvature, as well as MRI intensity and gradient features at multiple intra-/sub-cortical levels. We then fed these features into a two-stage cascaded classifier for automated lesion detection based on ensemble learning (**Figure 1**). The vertex-wise classification was designed to maximize sensitivity (i.e., *detecting a maximum number of lesional clusters*), whereas the subsequent cluster-wise classification aimed at improving specificity (i.e., *removing false positives while maintaining optimal sensitivity*). Using a 5-fold cross-validation, we evaluated our approach in 41 patients with histologically-verified FCD and 38 age- and sex-matched healthy controls. Notably, routine MRI was initially reported as unremarkable in 80% (33/41) of patients. Two experts, blinded to clinical information, independently segmented FCD lesions on co-registered T1w and FLAIR MRI. Inter-rater Dice agreement index was 0.91 ± 0.11.

RESULTS

At the first vertex-wise stage, the classifier detected all but 4 lesions (37/41 = 90% sensitivity). However, it also detected false positives (mean ± SD clusters: 25 ± 23 in patients; 7 ± 5 in controls). Subsequent cluster-wise classification yielded a sensitivity of 83% (34/41 clusters co-localized with the manual label), while it dramatically reduced the number of false positive clusters (4 ± 5). An example is shown in **Figure 2**. Specificity was 92%, with only one cluster in 3 healthy subjects.

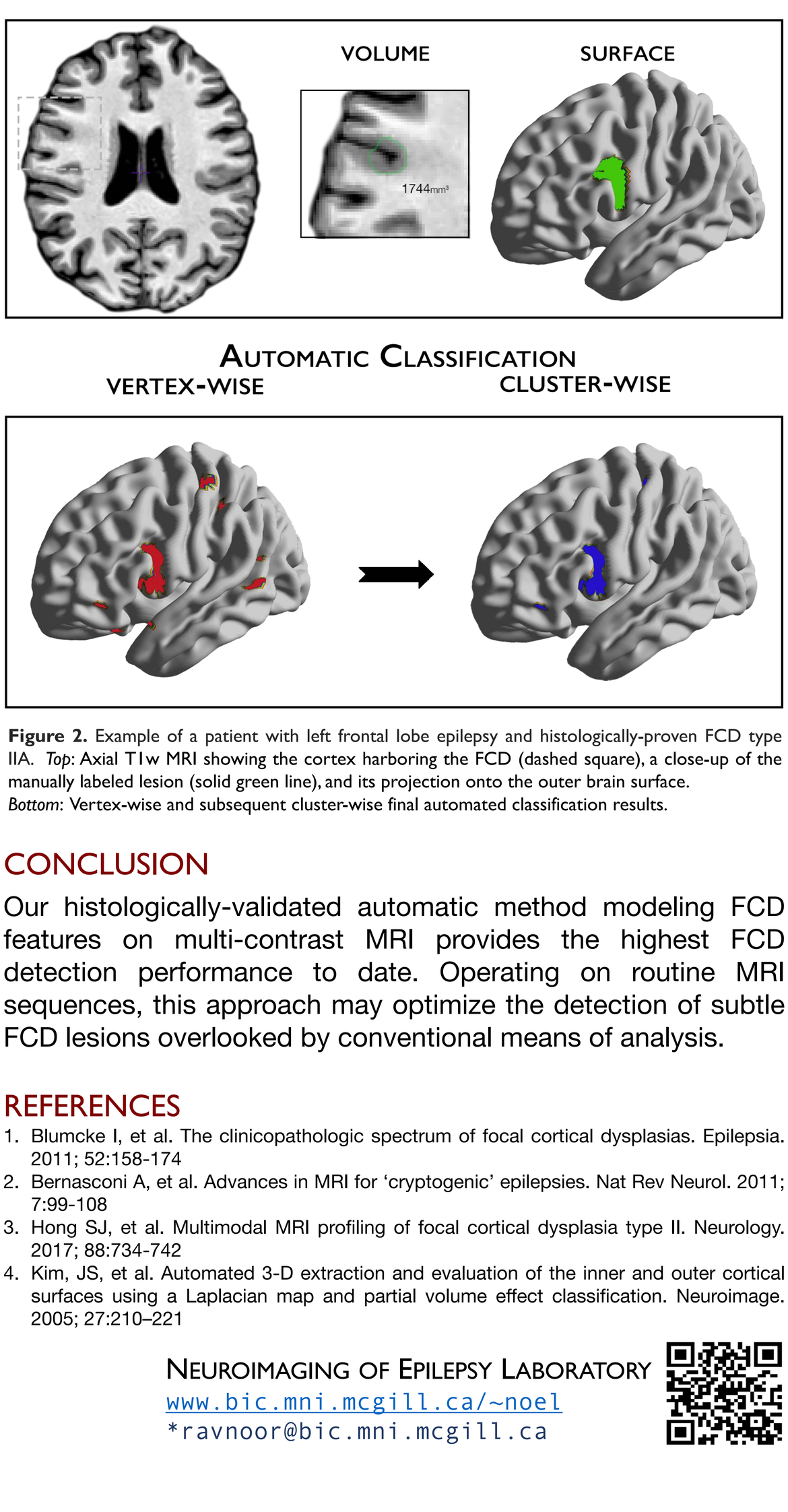

Figure 2. Example of a patient with left frontal lobe epilepsy and histologically-proven FCD type IIA. *Top*: Axial T1w MRI showing the cortex harboring the FCD (dashed square), a close-up of the manually labeled lesion (solid green line), and its projection onto the outer brain surface. *Bottom*: Vertex-wise and subsequent cluster-wise final automated classification results.

CONCLUSION

Our histologically-validated automatic method modeling FCD features on multi-contrast MRI provides the highest FCD detection performance to date. Operating on routine MRI sequences, this approach may optimize the detection of subtle FCD lesions overlooked by conventional means of analysis.

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