[Contribution of diffusion tensor magnetic resonance imaging to the diagnosis of focal cortical dysplasias].

[Article in Spanish]

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Abstract

INTRODUCTION:
Focal cortical dysplasias (FCD) are cortical malformations and, although they display typical characteristics in conventional magnetic resonance imaging (MRI), the precise determination of the epileptogenic zone remains a controversial issue. The less favourable progress during the postoperative period with respect to other symptomatic epilepsies could be explained by the existence of epileptogenic areas that do not show up in conventional MRI. Diffusion tensor imaging (DTI) is sensitive to subtle microstructural abnormalities, and fractional anisotropy, which is an indirect indicator, shows areas with reductions in the underlying white matter that go beyond the alterations detected with conventional MRI in isolated cases in previous works.

AIM:
In this study we analyse the characteristics of fractional anisotropy in a series of patients with FCD in order to evaluate the contribution made to diagnosis by MRI by DTI.

SUBJECTS AND METHODS:
Twenty-one controls and eleven patients with FCD that was visible in MRI were scanned, and clinical and imaging variables were both recorded. A visual analysis of the fractional anisotropy maps was conducted to search for asymmetries between hemispheres and biases in the clinical or structural MRI data.

RESULTS:
Two females and nine males, aged 30 ± 9.7 years took part in the study; time to progression of epilepsy: 22 ± 9.3 years; average frequency of the seizures: 3/month (range: 0.16-8/month). All of them showed inter-hemispheric asymmetries, which went beyond the structural limits of FCD in the case of 10 of the patients (90%). None of the controls displayed asymmetries in the fractional anisotropy. No significant relation was found with the variables that were compared.

CONCLUSIONS:
Further studies need to be conducted with larger numbers of patients in order to evaluate the usefulness of DTI in defining the location and extension of the epileptogenic zone in this population.