Structural and Metabolic Asymmetries in the Hippocampus Increase with the Duration of Epilepsy

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Abstract

Several lines of evidence suggest that hippocampal sclerosis in temporal lobe epilepsy is asymmetrically bilateral. The factors underlying such asymmetrical damage are relatively unexplored. This study correlates the asymmetry index of damage from hippocampal volumetry, relaxometry and NAA/(Cho+Cre) ratio with the duration of epilepsy. Results showed correlations between the degree of structural and metabolic asymmetries with the duration of seizure disorder.

Introduction

In human temporal lobe epilepsy (TLE), unilateral hippocampal sclerosis (HS) is a common finding. However, post-mortem quantified analysis of both hippocampi in patients with epilepsy have revealed cases of bilateral HS, mostly being asymmetric [1]. Quantitative hippocampal MRI studies in TLE often show unilateral atrophy, prolonged T2 relaxation times and decreased NAA/(Cho+Cre) ratios with different degrees of asymmetry [2, 3]. However, while previous reports [4] have suggested a variety of factors that may be associated with the presence of hippocampal atrophy, including a history of complex or prolonged febrile seizures, age at seizure onset, and epilepsy duration the clinical correlations that may determine the course of development of pathological asymmetries are relatively undefined. In order to study the relation between the hippocampal asymmetry index (AI) and the duration of symptomatic TLE, we performed a cross-sectional study where the AI of hippocampal volumes (HCVOL), T2-relaxometry (HCT2) and NAA/(Cho+Cre) profiles (HCSI) were correlated with the duration of the seizure disorder in years, in a group of patients under pre-surgical evaluation.

Methods

54 chronic adult TLE patients and 33 age-matched controls were included in the study. Patients with lesionsal pathology and heterotopic abnormalities were excluded. Duration of epilepsy for each patient was calculated from the beginning of the repetitive episodes of spontaneous seizures unprovoked by an acute illness. Data was obtained by reviewing the medical records from the local surgery for epilepsy programme and calculated accordingly to the formula [5]:

$$\text{Duration (years) = age at neuro-MRI - age at development of spontaneous seizures}$$

All MRI images were obtained on a 1.5T Signa CV/i-NV/i (GE). Quantitative MR data was acquired and processed as described [6]. AI for HCVOL, HCT2 and HCSI were calculated using the formula [7], where R refers to the right side and L to the left:

$$\text{AI} = 100 \times \left( \frac{R-L}{R+L} \right)$$

Results

The mean HCVOL, HCT2 and HCSI ipsilateral to the seizure focus differed significantly between controls and TLE patients. As shown in Figure, the longer the duration of epilepsy, the greater are the structural and metabolic asymmetries in-between hippocampi.

Discussion

Although it does not discard the possibility that a certain degree of unilateral damage can be present at the beginning of the disease, this cross-sectional quantitative multimodal analysis of hippocampal pathology suggests that asymmetrical damage progresses over time.

References


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