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Temporal Lobe Epilepsy, Depression, and Hippocampal Volume

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Abstract

Objective—To evaluate the relationship between hippocampal volume loss, depression, and epilepsy.

Background—There is a significantly increased incidence of depression and suicide in patients with epilepsy. Both epilepsy and depression are associated with reduced hippocampal volumes, but it is uncertain whether patients with both conditions have greater atrophy than those with epilepsy alone. Previous studies used depression measures strongly weighted to current state, and did not necessarily assess the influence of chronic major depressive disorder ("trait"), which could have a greater impact on hippocampal volume.

Methods—Fifty-five epilepsy patients with complex partial seizures (CPS) confirmed by EEG had 3D-SPGR MRI scans for hippocampal volumetric analysis. Depression screening was performed with Beck Depression Inventory (BDI, 51 patients) and with the structured clinical inventory for DSM-IV (SCID, 34 patients). For the BDI, a score above 10 was considered mild to moderate, above 20 moderate to severe, and above 30 severe depression. MRI and clinical analysis were performed blinded to other data. Statistical analysis was performed with Systat (Point Richmond CA) using Student's t-test and analysis of variance.

Results—There was a significant interaction between depression detected on SCID, side of focus, and left hippocampal volume. Patients with a diagnosis of depression and a right temporal seizure focus had significantly lower left hippocampal volume. A similar trend for an effect of depression on right hippocampal volume in patients with a right temporal focus did not reach statistical significance.

Conclusions—Our results suggest that patients with right temporal lobe epilepsy and depression have hippocampal atrophy that cannot be explained by epilepsy alone.

Keywords

Epilepsy; Temporal lobe; Depression; Comorbidity; Hippocampus; SCID; BDI

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Introduction

The comorbidity of epilepsy and depression is being recognized increasingly (Kanner 2003). They occur together much more frequently than would be predicted by their frequency in the general population and the risk for suicide and depression is significantly higher in epilepsy patients (Lambert and Robertson 1999). The risk of epilepsy is also increased in patients with depression or with a previous history of attempted suicide (Hesdorffer et al 2000; 2006). However, it is not clear if this association is due to similar underlying pathological processes, psychosocial effects, or a combination.

The two diseases may share neuroanatomic localization. Neuroimaging studies in patients with depression as well as epilepsy show volume loss in the hippocampus. Depressed patients have hippocampal volume loss (Bremner et al 2000, MacQueen et al 2003), and bilateral reduction in hippocampal gray matter (Seidenberg et al 2005) that may be worse with more bouts of major depression (Sheline et al 1996). These findings parallel the hippocampal atrophy found in patients with temporal lobe epilepsy, who can have bilateral hippocampal atrophy, although more severe on the side of the focus. Moreover, volume loss ipsilateral to the seizure focus has been shown to be related to seizure frequency, generalized tonic-clonic seizures, and duration of epilepsy (Theodore et al 1999, Tasch et al 1999, Kalviainen et al 1998, Spanaki et al 2000, Fuerst et al 2003).

A previous study that evaluated volumetric hippocampal changes in comorbid epilepsy and depression used the hospital anxiety depression scale (HADS) which is a dynamic measure of depression, reflecting current mood but not necessarily underlying disease (Baxendale et al 2005). However, hippocampal volumetric changes may not be present at the first episode of new onset depression, and in contrast, may be found in patients with a past history of depression who are not depressed at the time of scan (MacQueen et al 2003). Thus, we used a measure of underlying depressive trait, the structured clinical inventory for DSM-IV (SCID) as well as the Beck Depression Inventory (BDI), in order to study the effect of depression on hippocampal volume in patients with temporal lobe epilepsy (TLE).

Methods

Patients

We studied fifty-five consecutively evaluated patients (19 women) with temporal lobe epilepsy (mean age: 35 years, range: 18 to 62 years) with complex partial seizures, with or without secondary generalization, established by ictal video-electroencephalography, who had been referred to the Clinical Epilepsy Section, NINDS, for uncontrolled seizures. Patients with extratemporal lobe epilepsy were excluded. Subjects underwent depression screening with Beck Depression Inventory (BDI, 51 patients) and with the structured clinical inventory for DSM-IV (SCID, 34 patients). Thirty-one patients had both. We categorized patients with bilateral temporal interictal spikes but with all ictal events arising from one side as having a unilateral focus. Bilateral patients had ictal events arising from both temporal lobes.

The SCID was performed by a psychiatrist (G.H.) and was used to define patients as having a lifetime diagnosis of major depression or not. BDI questionnaires were completed by patients. A score of above 10 was considered mild to moderate, above 20 moderate to severe, and above 30 severe depression. In the context of this study, we used ‘depressive trait’ to indicate a current or past history of depression, and ‘depressive state’ the presence of depression at the time of evaluation. The patients were taking a wide variety of antiepileptic drugs (AEDs); carbamazepine, lamotrigine, and levetiracetam were the most common. Others included valproic acid, zonisamide, gabapentin, and phenytoin. During the

course of the seizure disorder many other AEDs had been tried as well. At the time of evaluation, none was on antidepressant therapy, but intermittent exposure had occurred in the past. The study was approved by the National Institute of Neurological Disorders and Stroke Institutional Review Board.

Imaging

All patients underwent coronal 3D spoiled gradient recalled acquisition (matrix 256 × 256 with 0.9375 × 0.9375 × 1.5 mm slices, TE: 3, TR: 27, TA:20, FOV 240mm, Nex:1) MRI Scans (GE, Milwaukee, Wisconsin). These were loaded into a linux based system and hippocampi manually traced on each slice, then assembled into 3 dimensional volumes. The anterior head of the hippocampus was separated from the amygdala by the appearance of the inferior limb of the lateral ventricle. The hippocampus was traced superiorly around the choroidal fissure, curving laterally along the medial border of the temporal horn, and medially along the gray matter of the hippocampus up to its junction with the parahippocampal gyrus. It was traced posteriorly to include the entire tail upto and including the gyrus fasciola. We calculated a hippocampal asymmetry index (AI): 2* (ipsilateral volume − contralateral volume)/(ipsilateral volume + contralateral volume). The more negative the AI, the greater the relative hippocampal volume reduction ipsilateral to the epileptic focus.

Statistical analysis was performed with Systat (Systat Inc Point Richmond CA) using Student's T-test and analysis of variance to compare hippocampal volumes and depression state or trait. MRI and clinical analysis were performed blinded to other data. All MRI analysis was performed by the same reviewer to ensure reproducibility of methods.

Results

Twenty-seven patients had a left temporal focus (LTLE), 23 had a right temporal RTLE), and 5 had bitemporal foci. Left hippocampal volume was significantly lower in patients with LTLE than in those with RTLE ($p < 0.02$), and right hippocampal volume significantly lower in those with RTLE than LTLE ($p < 0.001$) (table 1). The mean AI did not differ significantly between LTLE (-0.32 ± 0.28) and RTLE patients (-0.22 ± 0.25) although there was a trend toward greater ipsilateral atrophy in LTLE patients. The patients with bitemporal foci had right hippocampal volume of 2528 ± 617, and left hippocampal volume of 2508 ± 623, consistent with bilateral atrophy.

Across the entire patient sample, patients with depression on SCID or BDI did not differ in right or left hippocampal volume, or AI, from those with negative depression assessments (table 1). There was no difference in BDI between RLTE (10.4 ± 10.3) and LTLE (11.9 ± 10.6) patients, or in the proportion depressed on SCID. One-way analysis of variance (ANOVA) for depression based on BDI compared to right (F-ratio 0.129, $p=0.721$) or left (F-ratio 0.710, $p=0.403$) hippocampal volumes showed no relationship. Severity of depression based on BDI did not show a relation to hippocampal volumes. Patients with increased MRI signal intensity diagnostic of mesial temporal sclerosis (MTS) on qualitative reading did not have higher BDI, or higher likelihood of positive SCID. There were no significant differences in age at depression onset, duration of illness or number of episodes between patients with LTLE and RTLE.

Patients positive for depressive trait on SCID had significantly higher BDI (14.2 ± 12.7 versus 5.5 ± 5.4 $p<0.02$) than those negative on SCID. Five patients with current depression on SCID had BDI of 22.8 ± 16.3, compared with 6.9 ± 6.3 ($p <0.01$) for those with only a history of depression.

Across the entire sample, there were interaction effects for SCID and focus on hippocampal volume (left: f-ratio 4.37, $p<0.05$; right: f-ratio 3.73, $0.05<p<0.10$). RTLE patients who were depressed on SCID evaluation had significantly lower left hippocampal volumes than those who were not depressed (Spearman correlation coefficient −0.54) (table 2). There was a trend for depressed RTLE patients to have lower left hippocampal volume. Similar trends were seen when depression was measured by BDI, but this did not reach statistical significance. LTLE patients showed no differences (table 2).

There was no difference between men and women in right or left hippocampal volume (table 1). Five of 13 women tested, and 14 of 21 men, were depressed on SCID (chi-square 2.59: $p = 0.11$), but there was no difference in BDI. Women were over-represented among patients with LTLE (13/28) compared to RTLE (4/23) (chi-square 4.79: $p <0.02$). However, there were no interaction effects between gender and SCID, or gender and focus, on hippocampal volume. There was a weak inverse relationship between depression duration and left hippocampal volume ($r^2=.18$, f-ratio 2.95).

Discussion

We found significantly lower left hippocampal volume (and a strong trend for right hippocampal volume) in depressed compared to non-depressed patients with RTLE. In contrast, there was no effect in patients with LTLE. Our results parallel a previous report that found greater hippocampal symmetry associated with higher depression levels in the right MTS patients, suggesting a smaller left hippocampus, and thus greater bilateral atrophy associated with depression (Baxendale et al 2005). A study using several scales, including the BDI, found that right MTS was related to increased scores (Nees et al 2001). However, our study suggests that the underlying depressive trait, detected on SCID, as well as the current mood state, measured by BDI or Hospital Anxiety and Depression Scale (Baxendale et al 2005), may influence hippocampal volume.

The vagaries of sampling in a consecutively acquired clinical series might explain our finding a significant relation between hippocampal volume and depression in RTLE alone. However, we were unable to find any explanatory clinical parameters. Although the gender distribution differed, there were no differences in right or left hippocampal size between men and women, or any interactions between gender and focus. Overall, the proportion of depressed patients in RTLE and LTLE groups was similar.

We found about 40% of our patients had positive findings on SCID or BDI. The rate of reported depression in epilepsy ranges from 20–55%; in a community study, female gender and low socio-economic status were associated with increased depression risk (Ettinger et al 2004). In a series from several epilepsy centers that excluded surgery patients, 22% of an unselected cohort had a mood disorder (Jones et al 2005). In a large single center series collected over 11 years, 33 % of patients had current or past depression on clinical evaluation; there was no difference between temporal and extratemporal lobe epilepsy, or between patients with RTLE or LTLE (Adams et al 2008). Consistent with our results, MTS was not associated with increased risk for depression. Forty-four % of 37 patients with refractory TLE had a history of depression, associated with length of epilepsy, but not laterality or MTS (Briellman et al 2007). One study (Quiske et al 2000) not confirmed by subsequent work from the same group suggested an association of MTS with depression (Helmstaedter et al 2004).

Our finding of an association between depression and reduced left but not right-sided volume in the RTLE patients could be attributed to sample size. Moreover, RTLE patients already have significant right hippocampal atrophy that may make an additional effect of

depression hard to detect. Detection of the effect in RTLE but not LTLE is harder to explain, but, intriguingly, has been suggested by two other studies (Nees et al 2001, Baxendale et al 2005). Right temporal lobe and hemispheric resections have been associated with the development of post resection depression (Kohler et al 1999, Quigg et al 2003). Right temporal resections tend to be more extensive, often involving greater subcortical tissue loss than left temporal resections. However, more recent reports from a multicenter trial have not confirmed this association (Devinsky et al 2005). There may be some evidence for lateralized limbic affective processing. Patients with RTLE onset before age five had impaired recognition of facial emotion (McClelland et al 2006). In a study of contextual fear condition, right but not left hippocampus was activated (Alvarez et al 2008).

Our study has several limitations. The sample size was small, especially when divided by seizure focus laterality. We did not have a formal mechanism for establishing cumulative antidepressant exposure. Moreover, the wide variety of AEDs patients were taking at the time of the study, or had taken formerly, makes it extremely difficult to assess their potential effect on hippocampal volume.

Our findings should be seen in the context of the reported association of primary depression with reduced hippocampal volume; the left hippocampus may be more affected than the right (Bremner et al 2000, Macqueen et al 2003, Sheline et al 1996) Some investigators have failed to find an association, while other report that factors such as a history of child abuse might be important; suggestions that hypercortisolemia might lead to hippocampal atrophy, a process with potential relevance for epilepsy as well, have not been confirmed (Geuze et al 2005). Some data suggest patients with epilepsy have impaired dexamethasone supression of corticotropin releasing hormone induced cortisol and adrencorticotrophin release (Zobel et al 2004). Data from MRI and positron emission tomography studies in primary depressive disorders and well as epilepsy with depression suggest that volume loss and dysfunction may be widespread (Bromfield et al 1992, Victoroff 1994, Drevets et al 1999, Rajkowska et al 1999, Zetzsche et al 2006, Richardson et al 2007). It may be that the additional hippocampal volume loss, as well as more widespread findings, seen in patients with depression as well as epilepsy is the result, rather than the cause of the mood disorder.

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Table 1

Hippocampal Volumes in 55 Subjects

	Right Hippocampus (mean S.D.)	**Left Hippocampus mean S.D.)**
Right temporal focus (n=23)	2561 ± 840*	3069 ± 554**
Left temporal focus (n=27)	3403 ± 450*	2549 ± 788**
SCID: depressed (n-15)	2910 ± 684	2940 ± 606
SCID: not depressed (n=19)	3153 ± 691	2902 ± 906
BDI: depressed (n=21)	2997 ± 821	2789 ± 606
BDI: not depressed (n=30)	3024 ± 725	2861 ± 801
Men (n=36)	2957 ± 769	2731 ± 716
Women n=19)	3049 ± 717	2790 ± 779

* $p < 0.001$ two sample Student's t test

** $p < 0.02$ two sample Student's t test

Table 2

The Effect of Seizure Focus and Depression on hippocampal volume

Focus (N)	SCID (N)	Right Hippocampus	Left Hippocampus
Right (14)	Nondepressed (8)	2960 ± 905**	3425 ± 648*
	Depressed (6)	2261 ± 378**	2852 ± 250*
Left (20)	Nondepressed (12)	3294 ± 485	2522 ± 898
	Depressed (8)	3405 ± 438	2952 ± 811

comparing hippocampal volumes and depression

status in patients with right foci and left foci

* p <0.05, two sample Student's t test

** p <0.10, two sample Student's t test