

The Default-mode Network Is Selectively Altered in Temporal Lobe Epilepsy

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Introduction

Medial temporal lobe epilepsy (mTLE) is the most common type of human intractable epilepsy characterized by the substrate pathology of hippocampal sclerosis and complex partial seizure symptom associated with the consciousness impairment. Behavioral and previous imaging evidences both imply that the brain default-mode function may be related to the process of mTLE[1]. However, the alteration of the default-mode network (DMN), being as an intrinsic low-frequency fluctuation in the mTLE, is remaining unknown.

Material and methods

Twenty-two patients with right mTLE and the same number of patients with the bilateral mTLE participated in the current study with a control group of 27 healthy subjects. All groups of subjects underwent fMRI scans in a 1.5 Tesla MR scanner (GE-Signa) in the resting state. Two approaches of independent component analysis (ICA) and amplitude of low-frequency fluctuation (ALFF) analysis [2,3], were applied to investigate the alterations in the functional connectivity and ALFF of the DMN in the patients with mTLE. One-sample t-tests results of the ICs of DMN in 3 groups were yielded to observe the spatial changes of DMN in the patients ($p<0.05$, FDR correction). Two-sample t-tests between patients and controls were used to detect the functional changes of DMN (joint thresholds of height: $p<0.05$ and extent: $p<0.001$). In addition, A clinical variable of epilepsy duration was correlated to the values of z-scores in the ICA and the ALFF values in the patient groups ($P<0.05$).

Results

One-sample t-tests revealed that the spatial patterns of the DMN in the patients were changed with decreased areas in the dorsal medial prefrontal lobe (dMPFC), lateral and medial temporal lobes (MTLs) ipsilateral to the pathological sides (Fig 1). In the two-sample t-tests of ICA results, the dMPFC, bilateral MTLs and the ipsilateral lateral temporal lobes presented decreased functional connectivity in the patients. However, the two sample t-tests of ALFF manifested the decreases within the dMPFC and increases within the MTs and the lateral temporal lobes ipsilateral to the pathological sides. Surprisingly, the post cingulate cortex (PCC) showed consistent decreases across different methods and different groups in the patients (Fig. 2). The correlation analysis revealed that there were significant negative correlations between epilepsy durations and ALFF values within dMPFC in both groups of patients ($z=2.57$ and 2.71 in the patients with right and bilateral mTLE, both $P=0.004$. The Figures were not presented here).

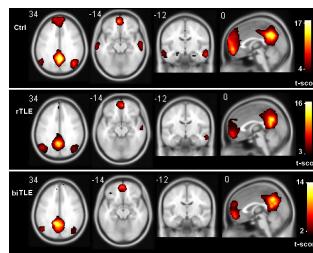


Fig.1. One-sample t-tests of the ICs of DMN
In each groups. Ctrl: Controls; rTLE: patients
with right mTLE, biTLE: patients with
bilateral mTLE. Left=Right

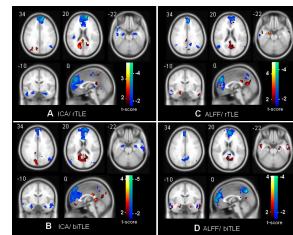


Fig. 2. Results of two-sample t-tests across
different methods and groups.
ICA(ALFF)/r(bi)TLE: Comparison of
ICA(ALFF) results between patients with
right(bilateral) mTLE and controls.

Conclusion

The consistent decreases in the functional connectivity and ALFF within the dMPFC of patients were suggested to relate to the functional impairments of consciousness and advanced cognitions such as automatisms and mental retardation. The decreased functional connectivity in the temporal structures implied the impaired memory function; while the increased ALFF represented the increased BOLD activation induced by epileptic activity in the epileptogenic regions. Whereas, the consistent increases in functional connectivity and ALFF in the PCC were interpreted to reflect the disturbances of the emotion and episodic memory retrieval, such as the psychic experience of déjà vu. In conclusion, our current findings supply a new insight into the pathophysiological mechanism of mTLE that the alteration of default-mode network may play a mediating role in the process of mTLE .

Reference:

[1] Engel J Jr. Neuroscientist 2001; 7: 340-52. [2] Greicius MD et al., Proc Natl Acad Sci USA. 2005; 101: 4637-42. [3] Zang YF et al., Brain Dev. 2007; 29: 83-91.