Forced thinking (FT) is a rare type of aura that refers to recurrent intrusive thoughts, ideas, or crowding of thoughts. Since Penfield and Jasper\(^1\) separated FT from psychic auras, FT has been regarded as an aura which is associated with frontal lobe epilepsy.\(^2\)\(^-\)\(^5\) However, FT has been also described as a psychic phenomenon arising from the temporal lobe in the literature.\(^6\)\(^-\)\(^8\)

We report on a patient who presented episodes of prolonged FT related to simple partial status epilepticus of mesial temporal lobe origin; which was demonstrated by intracranial electroencephalography (EEG) and ictal positron emission tomography (PET).

**Case Report**

A 50-year-old, ambidextrous woman was referred for the management of intractable partial seizures which began at the age of 13. During the first several years, she reported experiencing recurrent episodes of flash-backs which she described as “very familiar scenes passing by me”. She also reported olfactory hallucinations which consisted of odd smells. At the age of 17, she started to have complex partial seizures (CPS) which were associated with different types of psychic auras such as déjà-vu, jamais-vu, and FT. These auras were experienced alone or in various combinations.

She also had experienced two prolonged episodes of continuous FT which lasted for several days after clusters of CPS. She recalled these episodes of FT as “I forgot something that I should do”. Despite systematic trials of antiepileptic drugs (AED), she was still having two to three episodes of CPS monthly, and she decided to receive epilepsy surgery.

She had no prior medical history, including febrile seizure. A bedside neurological examination was unremarkable. Neuropsychological test revealed an average level of intelligence, and her memory was average in visual and borderline in verbal modalities. Magnetic resonance imaging showed left hippocampal sclerosis (Figure 1).

Video-EEG monitoring disclosed interictal epileptiform discharges in the left temporal region with a maximum amplitude at the sphenoidal electrode (SP1). Two episodes of CPS without aura were recorded after AED withdrawal. These started with brief eye blinking, subtle limb automatisms, and body rocking. She then developed moaning, right facial tonic spasms, right arm dystonia, and left head and body rotations. The ictal EEGs showed diffuse attenuation of background activity before the clinical onset, followed by delayed rhythmic 5-6 Hz theta activity in the left temporal region, 15s and 28s after the clinical onset, respectively.

Ictal $^{99}$Tc-ethylcysteinate dimer single photon emission computed...
tomography showed increased blood flow in the left temporal and parietal lobes (Figure 1). We elected to place intracranial foramen ovale electrodes (FOE) under local anesthesia because the ictal EEG onset was diffuse and there was a significant delay of lateralizing discharges after the clinical ictal onset.

Interictal EEG showed frequent epileptiform discharges at the left FOE without any obvious changes in the scalp electrodes. After the placement of the FOE, she experienced 18 episodes of brief CPS overnight. Ictal EEG revealed that the onset in the left FOE consisting of rhythmic \(\beta\) activity with superimposed polyspikes for 6-10s. This evolved to higher amplitude rhythmic theta activity in the left temporal scalp electrodes, 10-20s later.

The CPS clustering was successfully terminated by the intravenous administration of lorazepam, and she resumed taking AEDs including lamotrigine 100 mg, valproate 300 mg, topiramate 150 mg, and pregabalin 75 mg bid. However, she started complaining of continuous FT. The content of FT, quoted as her saying, was “I have something that I must do”, and the similar content of FT had occurred as either habitual auras or prolonged aura episodes after

---

Figure 1. Neuroimaging findings of the patient. Fluid-attenuated inversion recovery oblique coronal brain magnetic resonance imaging shows left hippocampal sclerosis (A). Increased blood flow during a complex partial seizure is seen in the left temporal region on \(^{99m}\text{Tc}-\text{ethylcysteinate dimer single photon emission computed tomography} (B). \(^{18}\text{F}-\text{flurodeoxyglucose positron emission tomography during forced thinking} (C), and its statistical parametric mapping (D) shows focal hypermetabolism in the left mesial temporal region and the right cerebellum.

Figure 2. Electroencephalography during the aura status of forced thinking. Continuous 1-1.5 Hz polyspike and slow wave complexes are seen on the left foramen ovale electrodes. L/Fo, left foramen ovale; R/Fo, right foramen ovale. The numbers after L/Fo and R/Fo designate the number of contacts of the foramen ovale electrode: the smallest number indicates the contact located at the tip of the electrode.
CPS cluster. She well recognized that there was nothing that she actually should do, but she was not able to stop this line of thinking. These thoughts caused unnecessary anxiety. The EEG showed a continuous 1-1.5 Hz periodic pattern of polyspikes and slow wave complexes on the left FOE during the persistent FT (Figure 2). Her bedside neurological examination including memory function did not show any definite abnormality. She did not have any problem in daily life. The simple partial status persisted for five days.

Brain 18F-fluorodeoxyglucose PET during the episode of FT showed a focal hypermetabolism in the left mesial temporal region (Figure 1). The PET data was analyzed using MRico (http://www.sph.sc.edu/comd/rorden/mricro.html) and statistical parametric mapping 8 (SPM8) (http://www.fil.ion.ucl.ac.uk/spm). The patient’s PET images were initially transferred to the Analyze format with MRico and spatially normalized to our institutional PET template using the affine transformation of SPM8. Afterwards, the images were smoothed with a 12-mm FWHM Gaussian kernel and a statistical analysis was set up to compare the patient and the control subjects (n=32) with a two-sample t-test. We used a voxel height threshold at p=0.001 (uncorrected) and the minimal cluster size as 100 voxels. The SPM map was superimposed on the normalized T1 template using ANALYZE 5.0 (Biomedical Imaging Resource, Mayo Foundation, Rochester, MN, USA). This demonstrated increased metabolism in the left hippocampus, the left parahippocampal gyrus, and the right cerebellum (Figure 1). A Wada test with intracarotid injection of thiopental revealed bilateral language and right memory dominance (6 versus 0 out of 10 items). The patient underwent a left anterior temporal lobectomy. The pathology was consistent with hippocampus sclerosis. No further seizure including aura had developed during the 56 months follow-up after the surgery.

Discussion

FT is generally considered as a type of psychic auras. Other types of psychic auras comprise complex visual or auditory hallucination or illusion, memory flashback, illusion of familiarity, hallucination or illusion of self-image, and emotion; which are frequently evoked by temporal lobe stimulation or seizures.1,7 In contrast, FT has been regarded to be associated with FLE.1,5 However, some investigators described FT as a psychic phenomenon arising from the temporal lobe.6,8 Gloor et al. described a patient who had experienced aura of seeing or thinking of two comic strip characters.7 The EEG showed ictal onset in the left hippocampus, subsequent spread to the neighboring parahippocampus, and minimally to the amygdala but not to the temporal neocortex. This experience was also elicited by left amygdaloid stimulation without afterdischarge.

FT which originates in the frontal lobe may differ from FT of temporal lobe origin.2,4 The frontal lobe FT can be accompanied by an attempt to act on the thought (forced acts such as real behavior of the same content, vocalization, and gaze attraction), where it takes on colder and more ideational aspect, particularly more intentional, as thoughts that impose themselves and then need to find a way to materialize. On the while, FT of the temporal lobe origin requires the limbic system for expression. Therefore, the content of the FT is set in a much more intense and vivid experiential, emotional, and affective context.6,7; however the content of FT originating in the temporal lobe may be vaguer expression.6 The FT occurred in our patient did not provoke any real act, but also did not involve prior memory.

Considering the content of FT in our patients, the FT may represent simply marked memory deficit due to mesial temporal dysfunction related to seizure activity. However, bedside neurological examination did not show any definite memory dysfunction. In addition, the patient herself well recognized that the FT did not represent a real situation. Ictal activity was confined to the left FOE, and a Wada test showed adequate functional reserve in the right temporal lobe. Unilateral mesial temporal dysfunction due to ictal activity would not produce such marked memory deficit in the context of ipsilateral hippocampal sclerosis.

There are reports on obsessive-compulsive (OC) symptoms in patients with epilepsy, or EEG abnormalities in patients with OC disorder, which demonstrated improvement in OC symptoms with AED treatment in some patients.9-12 However, these findings might reflect the comorbidity of epilepsy and OC disorder, and an effect on OC symptoms by AED treatment itself, rather than direct association with seizure activity, such as simple partial status.

Results of ictal PET and EEG using FOE strongly suggest that the mesial temporal region as an anatomical substrate generating ictal FT. In addition, prolonged FT can occur as a seizure manifestation.

Acknowledgement

We confirm that we have read the Journal’s position on ethical guidelines for scientific publication and affirm that this report is consistent with those guidelines. None of the authors has any conflict of interest to disclose.
References

1. Penfield W, Jasper H. Epilepsy and the Functional Anatomy of the Brain. London: Churchill Livingstone; 1954.
2. Bancaud J, Talairach J. Clinical semiology of frontal lobe seizures. Adv Neurol 1992;57:3-58.
3. Chauvel P, Kliemann F, Vignal JP, Chodkiewicz JP, Talairach J, Bancaud J. The clinical signs and symptoms of frontal lobe seizures. Phenomenology and classification. Adv Neurol 1995;66:115-25; discussion 125-6.
4. Mendez MF, Cherrier MM, Perryman KM. Epileptic forced thinking from left frontal lesions. Neurology 1996;47:79-83.
5. Ward CD. Transient feelings of compulsion caused by hemispheric lesions: three cases. J Neurol Neurosurg Psychiatry 1988;51:266-8.
6. Brickner RM, Rosner AA, Munro R. Physiological aspects of the obsessive state. Psychosom Med 1940;2:369-83.
7. Gloor P, Olivier A, Quesney LF, Andermann F, Horowitz S. The role of the limbic system in experiential phenomena of temporal lobe epilepsy. Ann Neurol 1982;12:129-44.
8. Hill D, Mitchell W. Epileptic anamnesis. Folia Psychiatr Neurol Neurochir Neerl 1953;56:718-25.
9. Jenike MA, Brotman AW. The EEG in obsessive-compulsive disorder. J Clin Psychiatry 1984;45:122-4.
10. Ketel PA, Marks IM. Neurological factors in obsessive compulsive disorder. Two case reports and a review of the literature. Br J Psychiatry 1986;149:315-9.
11. Koopowitz LF, Berk M. Response of obsessive compulsive disorder to carbamazepine in two patients with comorbid epilepsy. Ann Clin Psychiatry 1997;9:171-3.
12. Kroll L, Drummond LM. Temporal lobe epilepsy and obsessive-compulsive symptoms. J Nerv Ment Dis 1993;181:457-8.