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Case report

Temporal plus epilepsies: Electrophysiology studied with interictal magnetoencephalography and intracranial video-EEG monitoring

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1. Introduction

Temporal plus epilepsies (TPE) are rare and there are few sporadic reports on this topic.[1–3] Clinically, TPE and temporal lobe epilepsy (TLE) are often difficult to differentiate simply by using only general clinical features and MRI data.[2,3] Ryvlin et al. suggested that the presence of early ictal signs and symptoms implying the initial involvement of the perisylvian region, the orbito-frontal cortex, or the temporo-parieto-occipital (TPO) junction should heighten suspicion of TPE.[1] Some TPE patients, who were misdiagnosed as TLE, usually continued to suffer from seizures after temporal lobectomy surgical procedure. Thus, successfully identifying TPE patients may lead to more effective corticectomy surgical procedures and improve surgical outcomes. In the previous literature,[2] ictal scalp electroencephalographic (EEG) findings have shown the differences between TPE and TLE, but the pathogenesis and intracranial electrophysiological characteristics of TPE are still not fully understood.[1,2]

The ictal electrocorticographic (ECoG) recorded by intracranial video-EEG (iVEEG) monitoring is considered to be a golden standard for diagnosis and location of the epileptogenic zone.[1] IVEEG, as an invasive technique, has also been used to study the spatiotemporal characteristics of intracranial epileptiform activities. Recently interictal MEG, as a non-invasive technique, has been reported to provide critical information in the placement of intracranial electrodes for nonlesional epilepsy cases.[4] Compared to iVEEG, interictal MEG is a relatively new technique which detects magnetic fields generated by cortical neuronal activity in a very high spatiotemporal resolution on the order of millisecond and is well suited to investigate the electrophysiological characteristics of TPE.

In this report, we present a TPE case and study the electrophysiological characteristics of TPE. In addition, we evaluate the value of interictal MEG and iVEEG (ictal and interictal) in the diagnosis and understanding of TPE.

1.1. Patient

The patient was a 36-year-old, right-handed male with a 13-year history of seizures. There was no past history of febrile seizures, significant head trauma or intracranial infection. The symptoms of his seizures included auditory hallucinations which lasted about 10–35 s and were followed by a blank stare, repetitive involuntary movements of his hands, and automatisms such as lip-smacking or swallowing. Each of his seizures would usually last between 3 and 5 min and the patient was unconscious during the seizure. After the seizure, the patient gradually regained consciousness but he would experience an episode of dysphoria in 10–20 min. Occasionally, his seizures evolved further into generalized tonic–clonic seizure. The characteristics of the ictal events were consistently described by multiple witnesses and two neurologists (ZR and ZHT). The patient failed to respond to antiepileptic drugs (AEDs) including carbamazepine, phenytoin, and valproic acid. The frequency of his seizures varied from 2 times weekly to 5 times per day, even when he was given the polytherapy (Valproic acid 1200 mg/day and Carbamazeping 600 mg/day). Although his neurological and general physical examinations were unremarkable, the patient showed obvious memory decline over the last two years. The Wada test showed left hemispheric dominance for language and bilateral memory activity.

The anatomical magnetic resonance imaging (MRI) was acquired using a high resolution 3.0 Tesla MRI machine (GE Sigma scanner, GE Healthcare, Milwaukee, WI, USA). The patient's anatomical MRI demonstrated the cerebromalacia in

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the right TPO junction. The patient was monitored by the VEEG and three episodes of spontaneous seizures were captured and recorded. The patient had either a simple partial seizure or two complex partial seizures during the episode. The complex partial seizures further led to generalized tonic–clonic seizures. Both the interictal and ictal epileptic discharges were analyzed by two neurologists (ZR and YL). According to the ictal VEEG recordings, we detected rhythmic spikes that arose from the right TPO junction and rapidly spread to the right hemisphere, and eventually to the whole cortex. The duration of each seizure was about 3–5 min. According to the interictal VEEG, we found single spike-slow wave complexes that intermittently released from the bilateral frontal lobe or the right TPO junction when he was awake and during all sleep stages.

Due to a lack of evidence in location of the epileptogenic foci, interictal MEG and iVEEG monitoring with subdual electrodes were ordered to assist in identifying the epileptogenic foci and therefore develop a thorough surgical plan for this patient before his surgery in August of 2010.

2. Methods

2.1. Magnetoencephalography (MEG)

Magnetoencephalography (MEG) data were recorded for 30 min using a 275 channel whole-head system (CTF VSM MedTech Systems Inc., Coquitlam, BC, Canada) in a magnetically shield room (MSR) (Vacuum-Schmelze, Hanau, Germany) that was designed to reduce environmental magnetic noise. To increase the likelihood of capturing spike events, the patient was sleep deprived the night prior to the MEG recording. The head position related to the sensor arrays was measured using three coils affixed to the nasion and preauricular points before the MEG recording. We recorded 15 epochs (120 s/epoch) of spontaneous brain activities using MEG. No seizure occurred during the MEG recording. Synthetic aperture magnetometry (SAM) with excess kurtosis (g2) and conventional Equivalent Current Dipole (ECD) were used to analyze the MEG data.[4]

2.2. Intracranial video-EEG (iVEEG) with subdural electrodes

In August of 2010, subdural grid electrodes were implanted in the subdural space to localize the epileptogenic foci. A 128 channel Bio-Logic digital VEEG system (Natus medical Inc., San Carlos, CA, USA) with subdural electrodes was used to continuously monitor the electrical activity of the brain. According to the results from MRI, VEEG, MEG, and seizure semiology, the neurosurgeon performed a right modified pterional approach and a left mini frontal craniotomy to place three grid electrodes with a total of 48 electrode plates on the cortical surface where the suspected epileptogenic zones could be, including the right anterior temporal lobe (ATL), the left anterior frontal lobe (AFL), and the right TPO junction (see Fig. 1A). Every electrode plate was stitched to the edge of dura mater to avoid movement of electrodes after placement. The patient was monitored by iVEEG immediately after the placement of electrodes.

3. Result

During the interictal MEG examination, frequent spike discharges were recorded. Both SAM (g2) and ECD localized interictal activity in the right ATL and the right TPO junction, defined as the irritative zones. The epileptic discharges simultaneously arose from the right ATL and the right TPO junction without any time lag (see Fig. 1B).

During iVEEG monitoring, three typical seizures and a few interictal activity were recorded. Most of the interictal epileptic discharges were recorded from the right anterior temporal electrodes which attached to the right ATL. The ictal period of ECoG recordings exhibited a complex epileptogenic network. Initially, high-amplitude bursting spikes were raised from and confined within the right ATL. Then, the amplitude of ictal spikes increased gradually. The ictal spikes lasted about 30–45 s and stopped within the right ATL. After several seconds, new rhythmic spike-slow wave complexes arose from the right TPO junction and widely spread to the right temporal lobe and other distant regions. The widely propagated epileptiform spikes lasted about 50–120 s and terminated within the right occipital lobe (see Fig. 2). The results from the ictal ECoG suggested sequential epileptic activity which first started and ended within the right ATL and then after couple of seconds arose from the right TPO junction and propagated widely to the right temporal lobe and other distant regions, and at last ending within the right occipital lobe. This relatively long time lag between the two regions made it more difficult to determine whether the right TPO junction should be included in the epileptogenic zones[5,6] as the other ictal onset zone beside the right ATL. The MEG results showed that the epileptic discharges simultaneously arose from the right ATL and the right TPO junction without any time lag, which indicated that epileptic activity was spatially independent in the right ATL and TPO junction. Taking the interictal MEG results into consideration, the right TPO should be included in the epileptogenic zones. Therefore, we defined the right ATL and the right TPO junction as the epileptogenic zones.

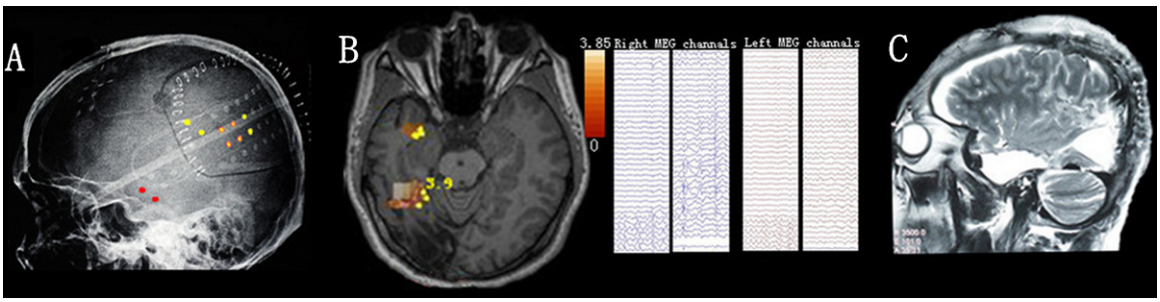

Fig. 1. (A) Skull X-ray image obtained after placement of the intracranial electrodes over the right temporal lobe, the left anterior frontal lobe and the right TPO junction shows the location of the ictal onset zones from the results of iVEEG recordings in red and yellow dots. (B) The results from MEG SAM (g2) (red) and ECD (yellow) were superimposed onto the patient's anatomical MRI. The color bar shows the color scale of the corresponding kurtosis value. The waveforms of MEG channels are shown on the right. (C) The anatomical MRI of the patient after the surgery shows the resected regions including the right ATL (including partial hippocampus and amygdala) and the right TPO junction. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of the article.)

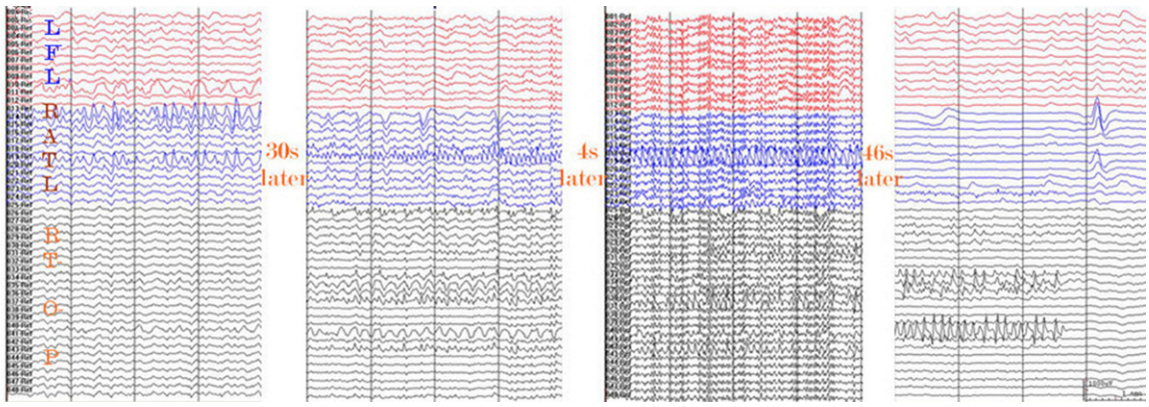

Fig. 2. The characteristics of the ictal ECoG were complex and involved two disconnected regions covering left frontal lobe marked by "LFL", right anterior temporal lobe marked by "RATL" and right temporo-parieto-occipital junction marked by "RTOP".

During the surgery, the patient underwent the epileptogenic zone resection of the right anterior temporal lobectomy (including partial hippocampus and amygdala) and the right TPO junction (see Fig. 1C). After surgery, the patient suffered from temporary anxiety disorder which gradually recovered in a week. He has been seizure-free for more than 18 months since his surgery.

4. Discussion

The interictal MEG data of this patient with TPE showed that the spike waves arose from both the right ATL and the right TPO junction, while VEEG localized epileptic discharges only in the right TPO junction. The interictal MEG results not only provided additional information that the right ATL was the suspected epileptogenic zone, but also improved the cortical coverage of the intracranial electrodes for the patient. According to the results from MRI, VEEG, MEG, and seizure semiology, we measured the ECoG including both the right temporal lobe and the right TPO junction. From the interictal and ictal ECoG results, only the right ATL was certainly defined as the epileptogenic zone. Due to the relatively long time lag between the right ATL and TPO junction, it is difficult to determine whether the right TPO junction should be included in the epileptogenic zones. But the interictal MEG indicated that epileptic activity was spatially independent in the right ATL and TPO junction areas. Thus, the right ATL and the right TPO junction were defined as the two epileptogenic zones were resected through surgery. The surgical outcome from our case has further proved that interictal MEG is a reliable and valuable tool for presurgical evaluation of the epileptogenic zones, in agreement with other studies.[4,7–9]

The limited number of subdural grid electrodes resulted in the incomplete cortical coverage between the right anterior ATL and the right TPO junction. The lack of this data may have added to the uncertainty about the nature of the relationship between the epileptic discharges observed in the right ATL and TPO junction areas. Based on available findings of ECoG, there was obvious time lag between the right ATL and the right TPO junction discharges. Due to the incomplete cortical coverage between the right anterior ATL and the right TPO junction, which limited spatial sampling, it is possible that the ictal spikes spread from the right ATL to the right TPO junction. On the other hand, there was no evidence of propagation of the interictal MEG dipoles over time between the right anterior ATL and the right TPO junction. The epileptogenic zones shall include all ictal onset zones and active irritative zones.[5,6] Taken together, we drew a conclusion that the patient's epileptogenic zones were situated in both the right ATL and the right TPO junction. According to diagnostic criteria,[1] the patient was diagnosed as TPE. Patients suffering from the TPE might not be seizure free when submitted to temporal lobe surgery.[3,10–12] Based on the pre-surgical evaluations from MRI, VEEG, MEG, seizure semiology, and ECoG, the resection regions included two epileptogenic zones in the right ATL (including partial hippocampus and amygdala) and the right TPO junction. The patient has been seizure-free since his surgery. However, we cannot be certain that the resection of the right TPO junction area was necessary to achieve complete seizure control due to the lack of evidence from the ictal ECoG.

Three episodes of seizure were captured during iVEEG monitoring. According to the interictal MEG results, both the right ATL and the right TPO junction should be defined as the epileptogenic zones. But the ictal ECoG exhibited a complex seizure-spread pattern. The epileptic discharges arose from and stopped within the right ATL, followed by some other new discharges which arose from the right TPO junction. The epileptic discharges which arose from the right ATL were prior to the right TPO junction, which formed a complex epileptic network during the same seizure.

In summary, the presurgical evaluation for our patient was done using multiple brain imaging techniques including MRI, VEEG, MEG, and iVEEG. We achieved a desirable surgical outcome. Our case shows that MEG, as a non-invasive technique, can provide us with additional and valuable information about the placements of the intracranial electrodes and the epileptogenic zones. Nevertheless, the epileptic network of TPE is so complex that the value of MEG for the TPE still needs to be further investigated.

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