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Surgical treatment of hypermotor seizures originating from the temporal lobe

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ABSTRACT

Purpose: To describe the characteristics of electroclinical manifestations in patients with hypermotor seizures (HMSs) originating from the temporal lobe.
Methods: We retrospectively reviewed the data of patients who underwent surgical treatments for seizure to identify patients with HMSs of temporal origin. We systematically reviewed patient seizure histories, imaging reports, video-EEG monitoring data, operative records and pathological findings.
Results: Eight of the 9 patients reported auras. The ictal behavior included marked agitation in 5 patients and mild agitation in 4 patients. All of the 9 patients exhibited stiffness or dystonia of the upper limb or contralateral limbs during ictus. Seven of the 9 patients completed intracranial recording and at least 3 seizures were recorded for each patient. The intracranial recordings showed ictal activity originating from mesial temporal lobe in 6 patients and the lateral temporal lobe in 1 patient. The time interval of ictal propagation from the temporal to frontal lobe was 15.0 ± 8.3 s. While the time interval from EEG origination to the beginning of hypermotor behavior was 21.0 ± 8.1 s. Brain MRIs revealed hippocampal sclerosis in 3, neoplastic lesion in 1, and normal images in the remaining 5 patients. Patients were followed for 1–5 years after the anterior temporal lobectomy; 7 patients remained seizure-free throughout follow-up.
Conclusion: Some HMSs can originate from the temporal lobe. In carefully selected patients, surgical resection may lead to good outcomes.

1. Introduction

Hypermotor seizures (HMSs) are seizures that are characterized by involvement of the body axis or proximal limb segments, resulting in large amplitude movements (such as rowing, kicking, or bicycling), and are commonly observed in frontal lobe epilepsy.[1] Recently, HMSs of extrafrontal origin have also been described, for example, in patients with temporal lobe,[2–4] insular lobe,[5–8] or even parietal lobe epilepsies.[9] However, the mechanisms by which extrafrontal areas activate complex behaviors observed in HMSs remain unclear. It appears that a complex anatomic and functional network may participate in HMS epileptogenesis. In this article, a number of electroclinical features of HMSs originating from the temporal lobe are described. These data may provide helpful information to understand the localization of the epileptogenic zones of temporal HMS patients and the complex mechanisms underlying special epileptic manifestations.

2. Patients and methods

2.1. Patients

We retrospectively reviewed the data of patients who received surgical treatments for epilepsy at the Comprehensive Epilepsy Center of Beijing between April 2001 and February 2012 to select those who met the following criteria:

1. The clinical feature of seizures were identified as HMSs according to an ictal video-EEG recording;
2. The epileptogenic zones were localized in the temporal lobes according to a presurgical evaluation;
3. The patients were seizure-free or rarely experienced seizures after surgical resection of epileptogenic zones.

Of the 1360 patients that were reviewed, 9 (0.7%) satisfied the inclusion criteria. We systematically reviewed the seizure histories, imaging reports, video-EEG monitoring data, operative records and pathological findings of these patients. This group consisted of 4 males and 5 females who were aged 15–31 years (mean age: 21.8 ± 5.0 years). All patients had intractable epilepsies

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that were resistant to the appropriate anticonvulsant medications. (Table 1)

2.2. Presurgical evaluation

2.2.1. MR imaging

Each patient underwent a standard MRI protocol that was performed using a 1.5-T MR scanner (Siemens Magneton Vision; Siemens, Munich/Erlangen, Germany) and consisted of conventional spin-echo T1-weighted axial, sagittal, coronal and T2-weighted axial sequences (section thickness: 5 mm, image gaps: 1 mm). Additionally, 5-mm-thick Fluid Attenuated Inversion Recovery images were obtained such that axial and coronal sections were perpendicular to the long axis of the hippocampus.

2.2.2. Video-EEG monitoring and seizure semiology

Each patient underwent interictal/ictal scalp EEGs that were recorded using a video-EEG monitoring system (Micromed; Treviso, Italy) with electrodes placed according to the international 10–20 system. The duration of video-EEG monitoring ranged from 2 to 15 days, and at least 3 habitual seizures were recorded for each patient. The major ictal manifestations were categorized according to the International League Against Epilepsy (ILAE) classification and the Rheims' classification systems.[1,10] Ictal manifestations were categorized as: (1) marked agitation including body rocking, kicking or boxing behavior while sitting upright, or (2) mild agitation characterized by horizontal movement or rotation of the trunk and pelvis.

2.2.3. Intracranial EEG monitoring

Intracranial EEG monitoring was performed in 7 of the 9 patients to confirm temporal lobe seizure origination. In these patients, the frontal lobe and temporal lobe were exposed during the procedure. Electrodes were implanted in both the temporal and frontal lobe. The placement of grid electrodes in the frontal lobe was guided by noninvasive exams and intraoperative electrocorticography (ECoG). In most cases, additional strip/grid electrodes were implanted to cover the cingulate gyrus and/or the orbitofrontal cortex. The mesial structure was also covered to identify possible involvement of the temporal lobe in seizure origination (Fig. 1). At least three habitual seizures were recorded for each patient during intracranial EEG monitoring. We reviewed the traits of each seizure including the ictal pattern, time interval of propagation, origination behavior, etc.

2.3. Surgery and pathological diagnosis

Classical anterior temporal lobectomy was performed in all nine patients (4 cm in the left anterior temporal lobe and 5 cm in the right anterior temporal lobe). All patients were followed postoperatively for more than 1 year. All tissue sections were routinely processed to obtain detailed pathological diagnoses.[11] Histopathological diagnoses were made using Palmini et al.'s terminology and classification of cortical dysplasias were made according to the World Health Organization Classification of Tumors of the Central Nervous System by at least two neuropathologists.[12,13]

3. Results

The characteristics of all 9 patients are summarized in Table 1. None of our patients had a family history of nocturnal seizure. One patient experienced seizure attacks dominantly in the evening, 2 patients experienced mainly daytime seizures, and 6 patients experienced seizures equally throughout the daytime and evening.

Table 1
Patient demographic characteristics and clinical manifestations.

Patients No.	Gender	Age		MRI abnormality	Seizure manifestation				Surgical removal	Histology	Outcome	
		Seizure onset	Surgery		Dominating seizure occurring	Aura	Subtype	Manifestation before HMS	Accompanied symptom			Seizure-free	Follow-up (month)
1	M	13	31	–	Daytime+evening	Feeling of blankness	Marked	Staring, hemifacial twitch	L-upper arm rigidity, hemifacial twitch	R–T	HS	N	12
2	M	4	22	–	Daytime+evening	–	Mild	Hand automatism	L-upper arm rigidity	R–T	HS	Y	12
3	F	3	18	–	Evening	Fear, nervousness	Marked	Scared facial expression, crying	L-limbs rigidity	R–T	FCD II b	Y	24
4	F	3	21	–	Daytime+evening	Fear	Marked	Opening eyes, arm rigidity	R-upper arm rigidity	L–T	FCD II a	Y	24
5	M	9	23	HS	Daytime	Rising gastric sensation	Marked	Opening eyes, hand automatism	R-upper arm rigidity	L–T	HS	Y	36
6	M	8	17	HS	Daytime+evening	Palpitation	Marked	Telling premonitions	L-upper arm rigidity	R–T	HS+FCD II a	Y	48
7	F	3	15	Neoplasm	Daytime+evening	Fear	Mild	Arm rigidity	R-upper arm rigidity	L–T	Ganglioglioma	Y	48
8	F	10	27	–	Daytime+evening	Rising gastric sensation	Mild	Opening eyes, hand automatism	R-upper arm rigidity	L–T	Heterotopia	N	12
9	F	3	22	HS	Daytime	Fear, palpitation	Mild	Hand automatism	R-upper arm dystonia	L–T	HS	Y	12

M: male, F: female, L: left, R: right, L-T: left temporal, R-T: right temporal, HS: hippocampal sclerosis, FCD: focal cortical dysplasia, Y: yes, N: no.