CASE REPORTS

SPECT findings during simple partial status epilepticus

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Abstract

Simple partial status epilepticus occurs more rarely than other forms of status epilepticus. This report describes two patients with temporal lobe epilepsy who experienced simple partial status epilepticus. Results show that basal ganglia and the mid-brain are activated during simple partial status epilepticus by blood perfusion single-photon emission computed tomography (SPECT) and subtraction ictal SPECT coregistered to MRI (SISCOM).

INTRODUCTION

Simple partial status epilepticus (SPSE) occurs more rarely than other forms of status epilepticus. This report describes two patients with temporal lobe epilepsy who experienced SPSE, and presents an assessment of the pathophysiology of SPSE inferred from results of blood perfusion single-photon emission computed tomography (SPECT) and subtraction ictal SPECT coregistered to MRI (SISCOM).

METHODS

SPECT and SISCOM procedures

All patients gave informed consent to SPECT imaging. During SPSE, 99mTc-labeled ethyl cysteinate dimer (ECD) was injected after several minutes from the clinical onset (Patient 1, 5 min; Patient 2, 3 min). The ictal event was confirmed by a trained epileptologist (K.S.). The same tracer was also injected to each patient during the interictal period. Ictal and interictal SPECT imaging were performed within 5 min after the injection using a triple-detector gamma camera equipped with low-energy fan-beam collimators (GCA-9300; Toshiba Medical Systems Corp., Japan). The imaging resolution was 10 mm full-width half-maximum after reconstruction. MRI images for SISCOM were acquired using a 1.5-T MRI scanner (Magnetom Vision; Siemens AG, Erlangen, Germany). Then SISCOM was performed using a commercially available software package (SISCOM software; Fujifilm RI Pharma Co. Ltd., Tokyo, Japan).

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Patient 1

Patient 1 was a 47-year-old Japanese woman born after an uncomplicated pregnancy. Her psychomotor development was normal. No family history of epilepsy was reported. She had experienced meningitis at the age of four years. At age 16, she had complex partial seizures (CPS) characterized by staring and unresponsiveness, subsequent to an aura of epigastric sensation. Scalp interictal EEG showed sporadic left anterior temporal spikes; MRI showed left hippocampal atrophy. Despite treatment with carbamazepine (CBZ), clobazam (CLB), and gabapentin, seizures were not controlled. She continued to have CPS weekly. The left temporal spikes on the EEG had become uncertain gradually. At the age of 46, topiramate was added to CBZ and CLB. Subsequently, the dosage was increased to 150 mg/day. Her CPS stopped. However, at age 47, she developed a new seizure type, which began with chest discomfort described as “an unpleasant sensation of constriction,” with subsequent rhythmic clonic jerks in the left arm and leg. Seizures lasted 1–2 hr in full consciousness without secondary generalization. They terminated...
spontaneously. Seizures occurred several times each day with the patient awake and during sleep. Seizures were always of the same pattern. She was admitted to our hospital for video-EEG monitoring. Interictally, sporadic small spikes were apparent, distributed over the right central and parietal regions. However, no change in EEG was found during seizures. To distinguish between epileptic seizures and psychogenic nonepileptic seizures, we performed ictal SPECT and SISCOM. Ictal SPECT revealed a focus of hyperperfusion in the right insular cortex and basal ganglia, in contrast to the left temporal hypoperfusion, which was observed interictally (Figure 1). Furthermore, SISCOM showed marked hyperperfusion clearly in the right putamen, pallidum and mid-brain (Figure 2). We diagnosed this episode as SPSE involving the right insular and basal ganglia. Although we discontinued TPM considering the possibility that it changed the ictal patterns of propagation, her SPSE never stopped. Treatment with higher dosages of phenytoin and valproate failed to control the seizures. Phenobarbital was of transient benefit. Zonisamide (ZNS) was dramatically effective, and stopped her seizures. She was administered ZNS 200 mg/day, CBZ 800 mg/day, and PB 65 mg/day. She subsequently had no CPS or SPSE.

Patient 2

Patient 2 was a 38-year-old mildly mentally handicapped Japanese woman. Her brother also had epileptic seizures. She experienced a febrile convulsion at the age of 1 year. At the age of 27, she experienced her first seizure consisting of dystonic posture of the upper left limb and loss of consciousness. She was treated with VPA, PB, and primidone, but SPS, including dystonic posture of the upper left limbs and CPS subsequent to the SPS, were not controlled. A scalp interictal EEG showed no definite paroxysmal activity; MRI showed right hippocampal atrophy. At the age of 34, monotherapy with CBZ was started. It markedly reduced her seizures. However, at the age of 37, a seizure of new type occurred. The seizures included arrested activity and speech with a vacuous expression, an intermittent clonic jerking of the upper left limb and an intermittent movement of scrubbing both hands. Although she was unable to respond to calling, she denied loss of consciousness. She was able to recall a word and a photograph that had been presented during the episode. The seizure lasted for several hours up to 1 day, and sometimes evolved into generalized tonic-clonic seizures. Although video-EEG monitoring showed no definite epileptiform discharge during seizures, ictal SPECT revealed a
focus of hyperperfusion in the left basal ganglia, in contrast to the right temporal hypoperfusion, which was observed interictally (Figure 3).

Furthermore, SISCOM revealed marked hyperperfusion in the lower left part of basal ganglia and mid-brain (Figure 4). We diagnosed this episode as SPSE involving the left basal ganglia and mid-brain, which rendered her akinetic, as with Parkinson syndrome. Lamotrigine was added to the CBZ; the dosage...
was increased gradually to 400 mg/day. Her seizure duration decreased from several hours to several minutes, but she still had one or two seizures each day. Subsequently PB was added. The seizure frequency decreased to several times a week.

**DISCUSSION**

In this study, we observed that basal ganglia and the mid-brain are activated during SPSE. Analyses using SISCOM clearly showed marked hyperperfusion in those regions (Figures 2 and 4).

In medial temporal lobe epilepsy, ictal dystonic posturing during CPS has been recognized. It is known to be related to the spread of ictal discharges to basal ganglia.3,4,6 Both our patients had unilateral hippocampal sclerosis. Patient 2 previously had ictal dystonic posturing during CPS. Although it differs from brief dystonic posturing (Patient 1, prolonged clonic jerking; Patient 2, akinesia, intermittent clonic jerking and a movement of scrubbing both hands), the results of SPECT and SISCOM show that the activation of basal ganglia and mid-brain might be related to movements that were observed in our patients.

Both our patients have a long-standing history of epilepsy followed by a recent occurrence of SPSE, which occurred after the suppression of conventional CPS by TPM (Patient 1) and by CBZ (Patient 2). Therefore, these drugs might change the ictal spread pattern, and ictal discharges might propagate to the contralateral basal ganglia and mid-brain over a long period.

Although we used ictal SPECT to investigate the epileptic activity during long-lasting seizures, ictal SPECT is generally performed to localize the ictal-onset zone for presurgical evaluation.5 Early ictal injections of a tracer minimize the problems of seizure propagation and nonlocalization because of an early switch from ictal hyperperfusion to postictal hypoperfusion.7 Injections were done after several minutes from the onset. Therefore, our results of hyperperfusion in the mid-brain and basal ganglia indicate a propagated zone rather than an ictal onset zone. Furthermore, animal reports have described that basal ganglia circuits cannot generate seizures. Moreover, they are unlikely to be involved in their initiation.2 Consequently, we consider that this area is related to propagated epileptic discharges originating in the cortex.

This study used analyses by ictal SPECT and by SISCOM. When using SISCOM, we must consider the possibility of normal variation among SPECT scans of single individuals. Brinkmann et al. reported high variance around the thalamus.

Figure 4. SISCOM of Patient 2. SISCOM images show marked hyperperfusion in the lower left part of basal ganglia and the mid-brain.
and hypothalamus. However, SISCOM clearly demonstrated the involvement of basal ganglia in relation to ictal dystonic posturing. The prolonged clonic jerking which our case showed is regarded as a kind of involuntary movement, which presents the possibility of involvement of basal ganglia and other subcortical regions. Therefore, we believe that our SISCOM findings are not a normal variance.

In conclusion, this study revealed that basal ganglia and the mid-brain are activated during SPSE, and that they might be related to prolonged involuntary movements in our patients.

**ACKNOWLEDGMENTS**

We confirm that we have read the Journal’s position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

**DISCLOSURE**

Conflict of interest to declare: Nil

**REFERENCES**