SHORT REPORT

Ictal magnetoencephalographic discharges from elementary visual hallucinations of status epilepticus

M Oishi, H Otsubo, S Kameyama, M Wachi, K Tanaka, H Masuda, R Tanaka

Magnetoecephalography (MEG) analysis typically uses an equivalent current dipole (ECD) model overlaid onto magnetic resonance (MR) images to localise sources of intracranial activity. As magnetic fields are comparatively unaffected by the different electrical conductivities of the brain, cerebral spinal fluid (CSF), skull, and skin, MEG can accurately localise the sources of intraneuronal electric currents that contribute to extracranial magnetic fields. MEG provides unique information about epileptogenic zones reflected by interictal spikes in patients with partial epilepsy. Ictal MEG studies, however, are rare, as head movements interfere with the accuracy of MEG source localisation. In the few successful ictal studies, MEG showed clustered spike sources from ictal ECDs and from localised ictal and interictal ECDs in epileptogenic zones.

Patients may experience simple partial status epilepticus (SPSE) as visual hallucinations. Elementary visual hallucinations (EVHs) consist of geometric figures, simple shaped figures, or flashes. EVHs are common in partial temporal lobe seizures.

We describe a patient with SPSE manifested as EVHs, in whom we simultaneously recorded MEG and EEG during an EVH episode and after EVHs were controlled by drugs.

CASE REPORT

A 26 year old right handed man had complex partial seizures consisting of initial ictal blindness, headache, loss of consciousness, and at times clonic convulsion of the right upper limb since he was 8 years old. His episodes occurred a few times a year between age 8 and 24 and were controlled with valproic acid and zonisamide. His developmental milestones were normal, and he had no family history of seizures.

At 24 years of age, he had generalised tonic status epilepticus and was intensively treated with a barbiturate. Thereafter he frequently complained of having EVHs, which he described as “snowing on TV screen,” “flickering lights,” and “rotating multiple coloured balls” in the right upper quadrant visual field. At times, the EVHs continued for several days. Carbamazepine was started for the EVHs.

The neurological findings, including visual acuities and fields, were normal. MR images (MAGNEX Epipal 15; Shimadzu, Kyoto, Japan), consisting of 1.5T high resolution, 1 mm thin slices, showed no abnormalities. Long term video EEG showed sporadic spikes and medium amplitude rhythmic theta waves over the left middle and posterior temporal regions during EVHs.

SIMULTANEOUS MEG AND EEG

We recorded MEG and simultaneous EEG twice: during an episode of EVH and after the EVHs disappeared. For MEG we used a helmet shaped neuromagnetometer consisting of 204 planar-type gradiometers (Neuromag 204; 4D-Neuroimaging, Helsinki, Finland) in a magnetically shielded room. For EEG, we placed scalp electrodes according to the International 10–20 system. We collected both MEG and EEG data digitally at a 300 Hz sampling rate with a low pass filter of 130 Hz. We analysed the data off line with a bandpass filter of 3–45 Hz. For each MEG spike, we calculated the single ECD source using a spherical model. We evaluated the dipole moments of the MEG spike sources and overlaid the ECDs, with respect to three anatomical fiducial points, onto MR images. We used the Mann-Whitney U test for statistical analysis and set the level of significance at p<0.05.

During the first MEG and EEG recording, the patient complained of persistent EVHs. The EEG recordings showed intermittent, medium amplitude, rhythmic theta waves with sporadic spikes over the left temporal region (fig 1A). In contrast, the MEG results showed essentially continuous, periodic (2–2.5 Hz) spike complexes over the left temporal region that persisted throughout the 20 minutes of recording (fig 1B). These identical and consistent MEG epileptic discharges had three phases (fig 1C): an initial spike, a second spike, and a wave.

Abbreviations: MEG, magnetoecephalography; EVH, elementary visual hallucination; ECD, equivalent current dipole; SPSE, simple partial status epilepticus.
We randomly selected 20 ictal MEG discharges and analysed each phase by the ECD method with a goodness of fit greater than 80%. We localised 14 ECDs from the initial spikes, 8 from the second spikes, and 16 from the waves (table 1). ECDs of initial spikes spread medially, adjacent to the grey and white matter junction (fig 1D). ECDs of second spikes had the smallest distribution and the strongest average moment of all phases. All ECDs clustered over the middle to posterior portion of the left superior temporal region.

When carbamazepine was increased, the patient stopped experiencing EVHs. During the second 20 minute MEG and EEG recording period, the patient reported no EVHs. Both MEG and EEG detected eight interictal spike discharges. Unlike the ictal recordings, no continuous MEG spike complexes were recorded. However, as with the ictal spikes, the eight MEG spike discharges consisted of three successive phases. We localised six ECDs from the initial spikes, eight from the second spikes, and five from the waves (table 1). All interictal ECDs were in the left temporal region.

Intercitial and ictal MEG spike sources colocalised in the same left superior temporal region. However, the moments (mean (SD)) of the interictal second spikes (233.0 (63.9) nAm) and waves (169.1 (33.7) nAm) were significantly stronger than the moments of the ictal second spikes (128.7 (32.8) nAm) and waves (109.2 (55.7) nAm) (p<0.05).

**DISCUSSION**

To our knowledge, this is the first report of ictal MEG epileptiform discharges in the left superior temporal region during EVH of SPSE. Previous reports indicated that MEG spike sources, recorded in cortical myoclonus of epilepsia partialis continua, were localised in the precentral cortex. The jerk-locked back averaging method confirmed the appropriate latency between MEG spike sources along the central sulcus and corresponding myoclonic jerks. The clustered spike sources of the continuous periodic ictal MEG discharges recorded during EVH may function in the same way as those recorded in myoclonus, but have different manifestations because of their location near the optic pathway.

In a report on patients with epileptic visual auras, ictal scalp EEG showed tempo-opercital theta-delta waves, and intracranial EEG indicated beta waves or low voltage fast activities in the tempo-opercital region. In a case report of a patient who experienced an EVH manifestation of SPSE, scalp EEG correlated a sharp/slow wave with a single flash episode. In our case, interictal and ictal MEG spike sources colocalised in the same left superior temporal region, but the rare interictal MEG spikes, even though large, were not associated with EVHs. We suggest that the continuous and periodic property of the ictal MEG spike sources in the ictal symptomatic zone, close to the optic radiation in the left temporal region, probably provoked an epileptic EVH in the right upper quadrant of our patient’s visual field.

All ECD localisations for the continuous ictal MEG spike complexes overlapped in a small area in the left superior temporal region. Simultaneous ictal scalp EEG, however, showed blunt theta waves. Several explanations could clarify these differences between MEG and EEG findings. Firstly, sampling

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**Table 1** Results of equivalent current dipoles (ECDs) of MEG polyspike and waves

<table>
<thead>
<tr>
<th></th>
<th>Initial spikes</th>
<th>Second spikes</th>
<th>Third waves</th>
<th>Initial spikes</th>
<th>Second spikes</th>
<th>Third waves</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of ECDs</td>
<td>14</td>
<td>20</td>
<td>16</td>
<td>6</td>
<td>8</td>
<td>5</td>
</tr>
<tr>
<td>Moment (nAm)</td>
<td>95.3 (41.0)</td>
<td>128.7 (32.8)*</td>
<td>109.2 (55.7)*</td>
<td>145.2 (84.7)</td>
<td>233.0 (63.9)*</td>
<td>169.1 (33.7)*</td>
</tr>
<tr>
<td>Positions (mm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>X</td>
<td>-58.3 (9.0)</td>
<td>-61.9 (3.9)</td>
<td>-59.8 (6.8)</td>
<td>-57.7 (4.6)</td>
<td>-59.0 (3.9)</td>
<td>-57.5 (5.7)</td>
</tr>
<tr>
<td>Y</td>
<td>-12.6 (8.4)</td>
<td>-19.0 (2.3)</td>
<td>-18.7 (9.4)</td>
<td>-5.7 (10.7)</td>
<td>-8.0 (9.4)</td>
<td>-16.1 (6.5)</td>
</tr>
<tr>
<td>Z</td>
<td>44.0 (8.7)</td>
<td>54.5 (4.3)</td>
<td>55.0 (7.0)</td>
<td>47.2 (10.9)</td>
<td>53.6 (12.2)</td>
<td>51.9 (2.7)</td>
</tr>
</tbody>
</table>

* X, auricular line, positive right and negative left; Y, nasion-inion line, anterior positive and posterior negative; Z, from the centre toward the vertex of the head, positive superior and negative bottom; Values for the centre of the head (x, y, z) are all zero. * p<0.05. Data shown as mean (SD).
limitations of 10–20 scalp electrodes might prevent recording of the low amplitude discharges from tangential sources, and possibly existing radial sources, in the posterior portion of the superior temporal region midway between T3 and T5 scalp electrodes. High resistance conductors, consisting of brain, CSF, skull and skin, diminish lower spike discharges, and prevent them from reaching remote electrodes. Secondly, because those high resistance conductors have high frequency filtering effects, the shorter duration of the initial and second spikes within a very narrow area (less than 6 cm²) may not show up on the scalp EEG. Thirdly, MEG delineated generators only along the superior temporal sulcus. When the cortical surface that sources of electrical activity occupy is small, the attenuation on EEG is large. The epileptiform discharges in a limited area results in lower amplitude and slower frequency on the scalp EEG.

The average moments of interictal MEG spike sources were stronger than those of ictal MEG spike sources, and the interictal EEG recordings showed more prominent spikes and waves than ictal EEG recordings. Simultaneous MEG and electrocorticography study showed that the percentage of spikes detected by MEG was proportional to the area of electrocorticographic epileptic discharges in the neocortical area. The large interictal EEG spikes may have resulted from activation of an extensive, but non-ictal, cortical area.

EVHs are frequently associated with occipital seizure activity. However, primary visual auras are also common in posterior temporal EEG foci. EVH consists of geometric figures, simple shaped figures, or flashes, provoked from the primary visual cortex, the striate cortex, adjacent white matter, and the temporo-occipital region. In our patient, the history of complex partial seizures, including ictal blindness, clonic convulsion of the right arm, and loss of consciousness, indicates an epileptic neural network in the centro-temporo-occipital lobe. His persistent EVHs express the continuous epileptiform discharges that MEG defined within the lateral temporal/medial occipital epileptic network. Though the mechanism underlying ictal variability is uncertain, electrical hyperexcitability associated with seizures reverberates within the neural networks, which operate together and inextricably to culminate in the expression of seizures.

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