Eating epilepsy: Phenotype, MRI, SPECT and video-EEG observations

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KEYWORDS
Eating epilepsy; MRI; Perisylvian area; SPECT; Video-EEG

Summary
Background: Eating epilepsy is one of the rare forms of reflex epilepsy precipitated by eating. Previous studies have demonstrated lesions due to variable aetiology involving the temporolimbic and suprasylvian regions.

Objective: To study anatomical correlates of reflex eating epilepsy using multimodality investigations (MR imaging, video-EEG and SPECT).

Methodology: Six patients (M:F = 3:3; mean age: 20.7 ± 4.9 years) with eating epilepsy were subjected to MRI of brain, video-EEG studies and SPECT scan. These were correlated with phenotypic presentations.

Results: Among the five patients with ictal recording of eating epilepsy during video-EEG, semiology was characterized by behavioural arrest followed by either flexion or extension of trunk and neck and two patients had speech arrest and four had salivation from angle of mouth. Another patient had EEG changes during “thought about eating”. Four patients had perisylvian frontal lobe lesions and one had high frontal lesion on MRI. Ictal EEG (n = 6) showed ictal rhythmic slowing/fast activity in parieto-temporal (n = 2) or fronto-temporal (n = 4) regions with subsequent secondary generalization in three. Ictal and interictal SPECT imaging showed changes in frontal lobe (n = 1), anterior temporal lobe (n = 1), and parieto-insular region (n = 1) suggesting it to be seizure onset zone. Three of four patients with structural lesions in MRI had concordant ictal EEG and ictal SPECT changes.

Conclusion: Lesions near the perisylvian region might play a major role in eating epilepsy.

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Introduction

Eating epilepsy is characterized by seizures closely related to one or several parts of eating. Although described as eating epilepsy, seizures triggered by eating often occur in
patients who also have spontaneous seizures and are not now classified as a separate epileptic syndrome but as a seizure type in the most recent proposal (Engel, 2001). The unusually high figures had been reported from Sri Lanka (Senanayake, 1994). Earlier, thirteen cases of eating epilepsy were reported from south India (Nagaraja and Chand, 1984). Seizures with eating are typically of complex or simple partial type, almost always related to symptomatic localization-related epilepsy (Remillard et al., 1998). There are reports with different underlying etiologies like hypoxic ischaemic encephalopathy, cortical dysplasia, polymicrogyria causing eating epilepsy (Kishi et al., 1999; Loreto et al., 2000; Manyam et al., 2010). There are very few functional neuroimaging studies in patients with eating epilepsy (Blauwblomme et al., 2011; Loreto et al., 2000).

The aim of this study was to study the regions of the brain involved in patients with eating epilepsy by MR imaging (MRI), Single photon emission computed tomography (SPECT), and video-EEG observations in patients with eating epilepsy.

Patients and methods

Six patients with eating epilepsy were evaluated prospectively at a university teaching hospital, a major tertiary care referral centre for neuro-psychiatric disorders in south India. Approval from Institute Ethics Committee and informed written consent from patients or relatives was obtained. All the patients underwent detailed evaluation with clinical history, physical and neurological examination, and investigations with MRI of brain, video-EEG and SPECT. Patients underwent MRI of brain on a 3T MRI machine (Achieva, Philips) with standard epilepsy protocol. The MRI was evaluated independently by the investigators (SS, RDB).

Video-EEG was conducted in the epilepsy monitoring unit (EMU), clinical neurophysiology laboratory, Department of Neurology at our centre in all the patients. The recordings of the study were carried out in a semi sound proof laboratory in a standard laboratory setting. Scalp EEGs were recorded on 16-channel **“Galileo NT (EBN)”** machine, employing international 10–20 system of electrode placement using standard parameters and procedures e.g. High Filter — 70 Hz; Low Filter — 0.1 Hz; Recording time: 30 min; Sensitivity: 7 μV/mm; sampling rate: 256 Hz. The electrical impedance was kept below 5 KΩ. The reference electrodes of the monopolar montage were linked to earlobes. Surface EMG electrodes (filter band pass — 20 Hz to 2 KHz; Sweep speed — 10s/page) were placed on biceps or tibial anterior muscles. Video-EEG was visually analysed by the investigators (SS, PSC) to look for the clinical semiology, ictal and interictal EEG findings. Activation procedures included hyperventilation, photic stimulation (5–50 Hz for 5 s at a stretch with eyes open and closed) and after giving food in all during recording to precipitate seizures.

SPECT scans were performed on Symbia T6 True Point SPECT machine available at NIMHANS. Radiotracer used in this study was 99mTc-ECD (ethylenecysteinate dimer). During ictal and interictal studies, 20 mCi of freshly prepared 99mTc-ECD was injected intravenously followed by normal saline flush. Images of ictal and interictal studies were acquired, between 45 min and 1 h after radioisotope injection, using a dual-head gamma camera equipped with low energy high resolution collimators (Symbia T6; Siemens, Erlangen, Germany). All the patients underwent an identical protocol. Interictal SPECT was carried out in all the patients with seizure free interval of 24 h. Ictal SPECT was performed after inducing reflex epilepsy with food and radiotracer (ECO) was injected immediately at the onset of the seizures. Tracer was injected with mean interval of 12.7 s after onset of ictus with range of 10–15 s from the onset of clinical seizure. Video recording of ictal event was done in all patients during procedure. Interictal and ictal SPECT images were evaluated for evidence of focal hyperperfusion or hypoperfusion abnormality by visual analysis by Nuclear medicine specialist.

The data was entered in digital spread sheet.

Results

Six patients (M:F = 3:3; mean age: 20.7 ± 4.9 years) with eating epilepsy were recruited for the study. The age at onset ranged from 8 to 14 years (mean: 11.3 ± 2.16 years; median: 11.5 years). The mean duration of epilepsy was 8.1 ± 4.9 years (median: 7.5); range: 3–16 years. Two patients had mild mental retardation. One patient had history of febrile convulsions in childhood. Six (86%) patients had uncontrolled seizures despite polytherapy. Post-evaluation, one patient underwent lesionectomy and was seizure free post-operatively. Five patients had abnormal MRI of brain. Four patients underwent ictal SPECT studies while five patients had ictus during video-EEG recording. One patient (case 6) had epileptiform activity in EEG provoked due to “thinking of eating” during video-EEG recording.

All the patients had ictus during the middle of their eating except one patient who had seizures at beginning of meal. One of the patient’s seizures was precipitated while eating rice containing food only. All patients had arrest of activity followed by flexion/extension of neck and trunk. There was speech arrest in three and drooling from the angle of mouth in five. Interictal EEG was abnormal in four (57%) patients, which included localized sharp waves in three and generalized spike wave discharges in one. Definite ictal EEG changes (n = 5) showed ictal rhythmic slowing/fast activity in parieto-temporal (n = 2), fronto-temporal (n = 1), fronto-central (n = 1) or fronto-centro-temporal (n = 1) regions ipsilateral to the MRI lesion in 3 of them. In one of the patient (case 5), obvious ictal EEG changes was not noted due to muscle/movement artefacts except for probably focal slowing to theta range across right fronto-central region noted after modifying the filter settings. One patient (case 6) had increase in sharp wave discharges over left fronto-centro-temporal region while thinking of eating without obvious clinical attack.

The MRI of brain showed structural lesion in 5/6 (83.3%). The MRI diagnosis included cortical dysplasia (n = 1), infantile stroke (n = 1), calciﬁed healed neurocysticercosis (n = 1), and unexplained gliosis (n = 2). The lesions were located near the perisylvian frontal lobe (n = 4) high frontal lesion (n = 1). Two patients had only interictal SPECT and one of them showed hyperperfusion in left frontal lobe with normal MRI. Ictal and interictal SPECT in the other 3 patients showed hyperperfusion in the frontal (n = 1), temporal (n = 1), and
Table 1 Clinical data and results of multi-modal investigations of seven patients of eating epilepsy.

<table>
<thead>
<tr>
<th></th>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
<th>Case 4</th>
<th>Case 5</th>
<th>Case 6</th>
</tr>
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<tbody>
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<td>26</td>
<td>17</td>
<td>23</td>
<td>16</td>
<td>16</td>
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<tr>
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<td>F</td>
<td>M</td>
<td>M</td>
<td>F</td>
<td>F</td>
<td>M</td>
</tr>
<tr>
<td>Age at seizure onset</td>
<td>10</td>
<td>14</td>
<td>13</td>
<td>11</td>
<td>8</td>
<td>12</td>
</tr>
<tr>
<td>Seizure semiology</td>
<td>Neck extension, speech arrest, salivation</td>
<td>Neck and trunk extension, flexion of both LL, salivation</td>
<td>Forward Neck and trunk flexion with nose wiping</td>
<td>Speech arrest, salivation, opening of jaw</td>
<td>Forward Neck flexion, salivation</td>
<td>Forward Neck flexion, salivation, speech arrest</td>
</tr>
<tr>
<td>Trigger stimuli</td>
<td>Eating</td>
<td>Eating</td>
<td>Eating</td>
<td>Eating</td>
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<td>Eating</td>
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<td>Spontaneous seizures</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Seizure frequency</td>
<td>5–6/month</td>
<td>3–4/week</td>
<td>1–2/day</td>
<td>1–2/day</td>
<td>1–2/day</td>
<td>1–2/week</td>
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<td>Medications</td>
<td>Polytherapy</td>
<td>Polytherapy</td>
<td>Polytherapy</td>
<td>Polytherapy</td>
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<td>Neurological examination</td>
<td>No deficit</td>
<td>Mild mental retardation</td>
<td>No deficit</td>
<td>No deficit</td>
<td>No deficit</td>
<td>No deficit</td>
</tr>
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<td>MRI brain</td>
<td>Left fronto-parietal perisylvian cortical dysplasia</td>
<td>Right &gt; Left sylvian and suprasylvian gliosis</td>
<td>Right frontal and perisylvian gliosis</td>
<td>Left frontal calcified granuloma</td>
<td>Left frontal</td>
<td>Left frontal perisylvian gliosis</td>
</tr>
<tr>
<td>Interictal EEG</td>
<td>Left central theta waves</td>
<td>Left fronto-central sharp waves</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Left fronto central sharp waves</td>
</tr>
<tr>
<td>Ictal EEG</td>
<td>Left parieto-temporal ictal slowing</td>
<td>Right Fronto-centro-temporal beta fast activity</td>
<td>Right fronto-temporo central ictal slowing</td>
<td>Left fronto central ictal slowing</td>
<td>Focal slowing to theta range across right fronto-central region</td>
<td>Increased sharp waves across left fronto-centro-temporal region on thinking of eating</td>
</tr>
<tr>
<td>SPECT localization</td>
<td>Left fronto-parietal</td>
<td>Left hemispheric-posterior frontal/parietal</td>
<td>Normal (inter-ictal)</td>
<td>Left frontal</td>
<td>Right medial frontal</td>
<td>Left frontal (interictal)</td>
</tr>
</tbody>
</table>

F, female; M, male; NCC, neurocysticercosis; SPECT, single photon emission computed tomography.
parieto-insular \((n=1)\) regions as seizure onset zone. Three of four patients with structural lesions in MRI had concordant ictal EEG and ictal SPECT changes. One patient with bilateral structural lesion had right hemispheric ictal EEG discharges whereas SPECT showed left hemisphere seizure onset zone suggesting two independent loci operating at different time. The details are provided in Table 1. The details of MRI, EEG and SPECT studies of patient 1 are provided in Figs. 1 and 2.

Discussion

Demography

The mean age at onset of seizures was in childhood in the early part of 2nd decade \((5/6)\) in our study. There are reports of variable age at onset ranging from 4 to 48 years with mean of \(21.6 \pm 10.3\) years \(\text{(Remillard et al., 1998)}\).

Aetiology

It is reported that eating epilepsy was almost exclusively related to symptomatic focal epilepsy. There are reports with different underlying etiologies like hypoxic ischaemic encephalopathy, cortical dysplasia, polymicrogyria causing it \(\text{(Kishi et al., 1999; Loreto et al., 2000)}\). In the present study, \(5/6\) \((83.3\%)\) patients had structural lesion on MRI which included healed neurocysticercosis, cortical dysplasia, perinatal stroke and gliosis. Previously authors have documented aetiological heterogeneity and variable pathophysiological mechanisms which underlie the eating epilepsy \(\text{(Remillard et al., 1998)}\). In the present cohort, lesions were mostly around perisylvian area: frontal — 4, parietal — 1. The occurrence of o_percular and congenital bilateral perisylvian syndrome has been documented in eating epilepsy \(\text{(Kishi et al., 1999; Manyam et al., 2010; Mateos et al., 1995)}\).

Clinical attack

All the patients in this study had seizures at the middle of their meal suggesting chewing, swallowing or gastric distension as possible triggers. One patient had history of seizures within few minutes of initiating the meal suggesting probably a non-gustatory stimulus as trigger. Interestingly, one patient had seizures on eating any ‘rice-containing food’, indicating role of gustatory, olfactory or absorbed chemical substance (if any) as trigger. The same patient had bilateral
perisylvian frontal lesion on MRI whereas others had unilateral parietal \( n = 1 \) or frontal lesions \( n = 2 \). Some common pattern in semiology on video was recorded in five patients. All the patients had arrest of behavioural activity followed by either flexion or extension of trunk and neck and three patients had speech arrest and four had salivation from angle of mouth suggesting extra-temporal epilepsy. Such regional involvement might indicate that the final effector pathways were near face area, pharynx and neck in the frontal lobe. There were reports of temporal lobe semiology in eating epilepsy (Remillard et al., 1998).

**EEG changes**

Interictal abnormality was seen in 4/6 (57%) patients. In previous studies, 32% to 76% of patients have reported interictal EEG (Koul, 1991; Nagaraja and Chand, 1984). In previous study, ictal EEG had shown involvement of fronto-central and temporal regions (Remillard et al., 1998) similar to the finding of parietotemporal and frontocentral ictal onset zone on EEG.

**SPECT observations**

There are no studies of SPECT in eating epilepsy. In our study, hypoperfused areas on interictal scan correlated with structural lesion on MRI in 3/4 patients with abnormal MRI. Three patients with structural lesion on MRI and ictal SPECT had concordance. This finding further confirms the role of structural lesion in seizure onset zone in eating epilepsy patients. It was interesting to note that case 3 with bilateral lesion on MRI had ictal onset zones in right hemisphere on ictal EEG and left hemisphere on SPECT scan. It is likely that during video-EEG and SPECT scan different seizures were recorded with onset from either hemisphere.

Functional MRI had also demonstrated that the orbitofrontal and temporoparietal cortices are parts of the central neuronal networks processing food stimuli (Porubská et al., 2006). Recent study had identified interconnections between the gustatory cortex and insulo-hippocampal epileptogenic circuit to be involved in eating epilepsy by fMRI and intracranial EEG observations (Blauwblomme et al., 2011). In the present study, MRI showed structural lesion in perisylvian frontal area but

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**Figure 2**  Multi-modality investigation in patient 2: (A–C) MRI FLAIR axial view (B) and MRI T1W axial (A) and sagittal (C) view showed gliosis with volume loss in right > left sylvian and suprasylvian region; D,d; E,e; F,f: SPECT (axial, sagittal and coronal view) interictal scan showed hypoperfusion which was well perfused in ictal scan in left hemisphere — posterior frontal/parietal region. (F) Interictal EEG showed runs of theta waves across F3, C3. (G) During ictus, ictal EEG showed initial artefact followed rhythmic beta fast activity right fronto-centro-temporal region.
ictal EEG and ictal SPECT showed wider ictal onset zone involving temporal and parietal lobe. It is difficult to determine the exact ictal onset zone based on surface EEG or SPECT observations due to rapid seizure propagation (Van Paesschen, 2004).

Remillard et al. (1998) had studied 10 surgically cases of eating epilepsy and found two types of localization, temporal and opercular. In the temporal subgroup, it was proposed that context of the eating and meal is important. While in the opercular group, the type of food might play an important role. The thalamic sensory input to the damaged sensory cortex might have a role. Based on the present study involving 6 patients with eating epilepsy, it might be hypothesized that perisylvian region is an important zone for eating epilepsy. This was documented in this study based on clinical, MRI abnormalities, EEG changes and SPECT scan alterations. However except in one patient, this cohort did not undergo surgery. Nevertheless, there might be an overlap between the 2 seizure onset subgroups. The triggers of a seizure are usually stereotyped for each patient, which include gastric distension, chewing, swallowing, the passage of food along the oesophagus, specific chemical factors or the mere sight of food and other complex stimuli, such as movement and proprioceptive afferents of muscular origin, have been considered. Interestingly one of the patients in this cohort had documented seizure during the thinking about eating. Seizures with eating are typically of complex or simple partial type, almost always related to symptomatic localization-related epilepsy (Remillard et al., 1998). Similarly, the patients in this series also had localization related symptomatic (n = 5) and cryptogenic (n = 1) epilepsy.

To conclude, there might be an involvement of complex neuronal circuits around sylvian fissure (near area of face) responsible for eating epilepsy, one of the uncommonly encountered reflex epilepsy. Further studies with PET, simultaneous fMRI—EEG and possible intracranial recording (if clinically indicated) might provide better understanding.

Conflict of interest

None declared.

References


