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

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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.

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

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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 — 10 s/page) were placed on biceps or tibialis 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, Erlanagen, 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 (ECD) 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 $\pm$ 4.9 years) with eating epilepsy were recruited for the study. The age at onset ranged from 8 to 14 years (mean: 11.3 $\pm$ 2.16 years; median: 11.5 years). The mean duration of epilepsy was 8.1 $\pm$ 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 postoperatively. 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 either 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 hypoperfusion 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