Clinical commentary

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# Seizures triggered by eating: a rare form of reflex epilepsy

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**ABSTRACT** – Eating epilepsy is rare and comprises reflex seizures induced by food intake presenting with broad clinical manifestations. Despite this heterogeneity, a unique focal impaired awareness seizure semiology localizing to specific brain regions has been noted. Here, we present a case with video-EEG depicting this characteristic clinical presentation and its informative electrographic correlate. *[Published with video sequence*].

**Key words:** reflex epilepsies, eating epilepsy, semiology, video-EEG

Eating epilepsy (EE) is a rare form of reflex epilepsy with very frequent precipitation of seizures by eating (Rémillard *et al*., 1998). Although universally noted, a higher geographic frequency (approximately 25/1,000) has been reported from the Indian subcontinent raising interest in pathogenic relations to genetic (Senanayake, 1990a; Senanayake, 1990b) and ethnic factors (eating habits) (Nagaraja and Chand, 1984).

The roles of specific mechanical (gastric distension, chewing, swallowing, chemical factors, sight of food) and complex stimuli (movement and proprioceptive muscular afferents) during eating have been explored (Nagaraja and Chand, 1984) though not found to be definitive. Taken together, however, precipitating factors appear to be necessary (Reder and Wright, 1982) in conjunction with the sequence of eating a meal to provoke a seizure (Cirignotta *et al*., 1977).

Despite the heterogeneity of presentations, eating-induced seizures typically manifest with focal onset impaired awareness (complex partial) seizures. Most patients with this phenomenon have symptomatic focal epilepsies (Patel *et al*., 2013). A breadth of epileptic abnormalities has been described though cases can be largely compartmentalized to epileptiform activity arising from the inferomesial temporal structures in complex partial semiology and less commonly simple partial phenotypes from the suprasylvian region (Rémillard *et al*., 1998). The triggers for these groups also seem to vary, with suprasylvian origin cases being triggered by proprioceptive or somatosensory stimuli

*VIDEO ONLINE*

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(Rémillard *et al*., 1998). Despite this clustering, rare presentations of generalized, myoclonic, head drop and atonic cases have been described (Aguglia and Tinuper, 1983).

In this report, we describe a case of EE in which a video-EEG recording of an eating-induced seizure was recorded. The case highlights characteristic clinical features, emphasizes the usefulness of video-EEG monitoring in diagnosis and demonstrates the difficulties of treatment in these cases with the ultimate goal of alerting clinicians to this rare and often misdiagnosed condition.

## Case study

We present a 47-year-old man, an immigrant from South Asia, who had his first seizure at the age of 17. It was a generalised tonic-clonic seizure, but detailed semiological descriptions were not available. He experienced recurrent episodes until the age of 30 and was treated with sodium valproate and phenytoin.

After six years of being seizure-free, he suffered a seizure while having a meal. Recurrent seizure episodes subsequently occurred with an aura, lasting a few seconds, characterised by some sensation in his head which he has found very difficult to describe. The aura is followed by unresponsiveness and head version to the right side with some clonic head jerking. Nystagmus ensues while he continues chewing despite not taking food into his mouth. He remains unresponsive for about a minute or two. Then he coughs or vomits and recovers from his seizure. Typically, there are no post-ictal symptoms. Approximately >90% of these seizures occur with meals, usually dinner, with rare episodes occurring independent of meals. Seizure frequency, on average, was twice a week when he was assessed.

We did not find any specific risk factors for epilepsy such as febrile seizures in childhood, serious head injuries or CNS infections, perinatal insults, or family history of epilepsy.

At the time of video-EEG monitoring, his antiepileptic drug therapy included lamotrigine, sodium valproate, and oxcarbazepine. Previous trials of lacosamide, clobazam, phenytoin, levetiracetam and perampanel were aborted due to the lack of efficacy or side effects. His neurological examination was normal. Multiple routine EEGs and two MRI brain scans (1.5 Tesla and 3.0 Tesla) were normal (*figure 1A-D*) . EEG was performed using standard 10-20 electrodes. The PET scan showed mild hypometabolism in the left mesial temporal region (*figure 1E, F*).

His four-day video-EEG monitoring showed no interictal epileptiform discharges. Three focal-onset seizures were captured. Two seizures occurred in non-rapid eye movement sleep, a couple of hours apart, and lasted approximately two minutes. In these events, the patient was lying on his left side. He abruptly raises his head and some flicking movements are visible in his left hand. The EEG shows phasic muscle artefacts suggestive of chewing (oral automatisms). However, the face is not clearly visible on the video and the body is partially covered by a blanket. He then coughs and sits up with some dry retching. He then spits saliva and falls back to sleep. The ictal EEG activity noted is non-lateralising and is seen over both hemispheres at the same time. The rhythm is characterised by low-amplitude fast activity which gradually increases in amplitude and decreases in frequency.

The third typical seizure occurred during dinner. The video EEG shows him having a conversation with visitors while mixing his food with fingers of the right hand and chewing. Suddenly he goes quiet, stops mixing food, and stares blankly while continuing to chew. Then he repeatedly touches the head with left hand. Forty seconds after the clinical onset, horizontal nystagmus and subtle side-to-side head shaking is visible. This is followed by clonic head version to the right (one minute and 25 seconds from the clinical onset), transient generalised stiffening, and spontaneous seizure termination with a cough. The seizure lasted two minutes and 25 seconds. Later, the patient described experiencing his typical cephalic aura at the seizure onset. The EEG ictal onset was masked by the chewing artefact. Thirty-two seconds from the clinical onset, an evolving rhythmic 16-Hz activity with admixed sharp components becomes visible bilaterally with a left-sided emphasis. This ictal rhythm gradually evolves into a slower frequency with an increasing amplitude (*figure 2*). During the last 10 seconds of the seizure, repetitive 4-Hz sharp-wave discharges are seen over the left posterior quadrant (*see video sequence*).

Further treatment options including epilepsy surgery and vagus nerve stimulation were discussed at that stage, but the patient was subsequently lost to follow-up.

## Discussion

Our case highlights several characteristic aspects of EE. The onset of seizures in the adolescent setting and the patient's South Asian ancestry is typical. Our patient reported >90% of his seizures occurring with eating, particularly at dinner. He experienced focal onset impaired awareness seizures in keeping with the previous descriptions in the literature (Rémillard *et al*., 1998). The seizures were refractory to antiepileptic medications, an observation made by previous researchers (Nagaraja and Chand, 1984).

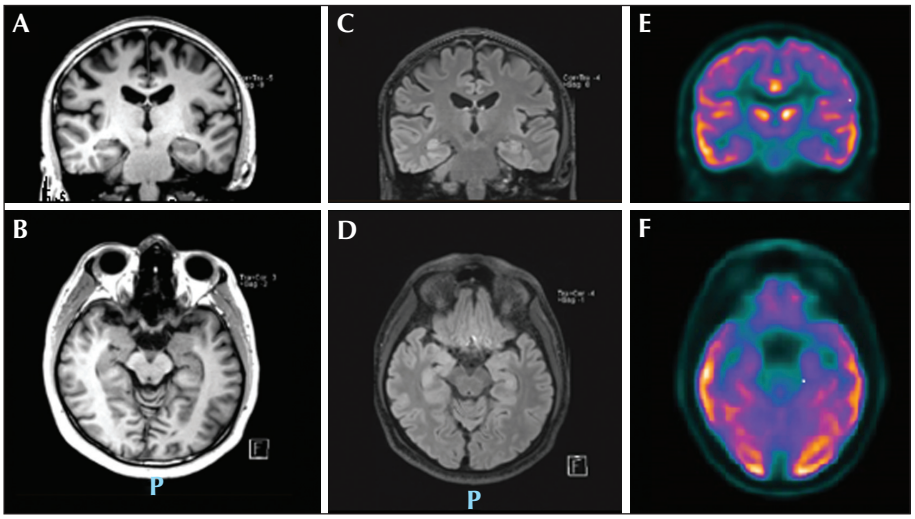


**Figure 1.** (A-D) MRI of the patient: T1 coronal (A), T1 axial (B), coronal FLAIR (C), axial FLAIR (D); MRI was normal and showed no cortical malformations or lesions. E, F) PET of the patient: coronal (E) and axial (F), showing subtle hypometabolism in the left mesial temporal region.

Eating epilepsy is a rare condition and the confirmation with video-EEG is even rarer in the literature. We were able to capture his typical seizure semiology with eating to establish the diagnosis.

Our investigations revealed some lateralising and localising information regarding the seizure onset zone. The PET scan demonstrated subtle hypometabolism in the left mesial frontal region. The ictal head version to the right may also indicate left-sided seizure onset, however, we should not disregard the fact that the head version occurred fairly late. The ictal EEG onset was masked by artefacts, but the rhythm later appeared well-developed on the left, perhaps favouring a left hemispheric onset. The localising value of the cephalic aura he experienced was poor and has been reported in seizures arising from several regions including the entorhinal cortex, amygdala, and temporal neocortex (Foldvary-Schaefer and Unnwongse, 2011). Early behavioural arrest witnessed in our patient is in favour of mesio-lateral or lateral temporal seizure onset (Maillard *et al*., 2004). Remillard *et al*. (1998) proposed two distinct syndromes based on seizure onset; temporolimbic and extralimbic. Our case most probably would fit into the temporolimbic group. We have not found a structural abnormality on MR imaging. However, given the adolescent onset of seizures and subtle PET abnormalities, underlying focal cortical dysplasia remains a possibility.

The presence of a family history has variably been reported, though not present in our case. The possibility of genetic susceptibility was first observed in nine families from Sri Lanka (Senanayake, 1990a), although food and eating habits were also hypothesized to contribute to such sibling clustering. Idiopathic temporal lobe seizures provoked by eating in a family of three women further supports a potential genetic link (Yacubian *et al*., 2004). Similarly, neurodevelopmental conditions (*e.g*., *MECP2* duplication, Rett syndrome) have also been reported in a small proportion of patients with eating-related seizures (Italiano *et al*., 2016).

In eating epilepsy, the relationship between seizures and food is consistent although a small proportion of seizures can occur at other times unrelated to meals (Rémillard *et al*., 1998). While seizures more commonly occur precipitously during meals, as noted in our patient's captured episode, cases of seizures being triggered just before, at the very beginning, towards the end or immediately after a meal are well documented (Ahuja *et al*., 1988). The most common presentation is, however, seizures while eating (approximately 88%) usually within five minutes of starting to eat (Senanayake, 1990a), as historically noted by our patient. Although the thought of food has been previously noted to trigger seizures (El Bouzidi *et al*., 2010), this was not the case in our patient. Focal impaired awareness seizures are most commonly noted in these cases, as seen in our case. However, detailed semiological descriptions are rare in the literature. Although generalised tonic-clonic seizures and simple focal seizures may occur in isolation, the coexistence of two or more seizure types and focal-onset seizures evolving into bilateral tonic-clonic seizures are often evident (Seneviratne, 2005).

The interictal EEG can be normal (over a quarter of reported cases) as mirrored by our case though focal slowing and epileptiform discharges predominate

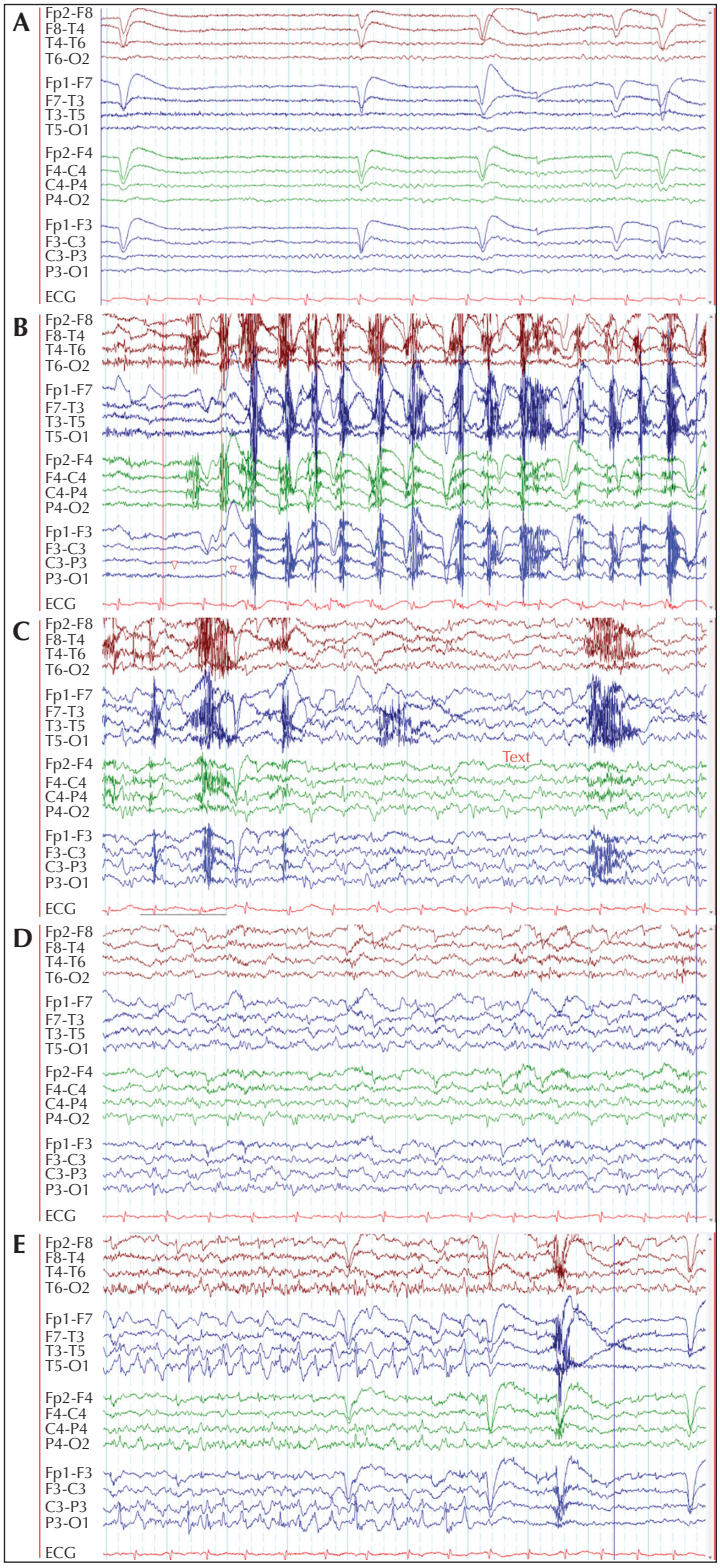


**Figure 2.** (A) Interictal EEG showing normal background activity. (B) Seizure onset occurs during eating; electrographic ictal onset is masked by the chewing artefact. (C) Once chewing movements cease, rhythmic 16-Hz activity with admixed sharp waves are visible bilaterally, but more prominent over the posterior head regions (left>right). (D) Evolving ictal rhythm with increasing amplitude left >right. (E) Seizure offset.

abnormalities detected (Rémillard *et al*., 1998). Epileptiform activity tends to lateralise and occur mainly over the temporal lobe (Senanayake, 1990a). Generalised epileptiform discharges and slowing have, however, been noted in approximately 10% of cases (Jagtap *et al*., 2016). Continuous video-EEG monitoring has been found by others to be useful in capturing the events considering the relatively frequent seizure presentation in these cases (Loreto *et al*., 2000).

The growing utility of imaging in reflex epilepsies has added diagnostic value in many cases while also enhancing knowledge on the likely underlying pathophysiology of EE. MR imaging has detected cortical malformations such as dysplasia, gliosis and polymicrogyria amongst others (Patel *et al*., 2013; Jagtap *et al*., 2016). Lesional epilepsies, including malformations of cortical development and hippocampal sclerosis, were found in 34% of cases in one series (Jagtap *et al*., 2016). Cases with ictal SPECT further demonstrate a mixture of localised perfusion changes corresponding to seizure onset zones within the frontal, temporal and parieto-insula regions (Patel *et al*., 2013). EEG-fMRI provides an opportunity to study the networks involved in the generation of reflex epilepsies with good spatio-temporal resolution. In a case involving a child with EE, EEG-fMRI revealed a widespread network involving mesial frontal, temporal, and parietal regions (Sandhya *et al*., 2014).

Antiepileptic drugs (AEDs) are required in the vast majority, with approximately half of the patients requiring polytherapy (Senanayake, 1990a). The most commonly used AEDs are phenytoin and carbamazepine, although clobazam has also been found useful (Aguglia and Tinuper, 1983). Despite best management efforts, EE can be drug-resistant in a substantial proportion, as seen in our case. Drug-resistant EE patients should be evaluated for epilepsy surgery and cases similar to ours may need stereo-EEG exploration. Vagus nerve stimulation appears to be efficacious in some patients with drug-resistant EE (Cukiert *et al*., 2010). As our patient is drug-resistant, epilepsy surgery and VNS therapy remain further treatment options.

We applied only the standard 10-20 electrode system in the EEG study, which is a limitation in retrospect. Extra electrodes such as anterior temporal (T1, T2), zygomatic, foramen ovale, and sphenoidal would have been more informative in localising the seizure onset and irritative zones.

In summary, we present a case of eating epilepsy highlighting typical features of this rare condition. We describe the seizure semiology in detail with EEG findings and emphasize the importance of video-EEG monitoring with provocation (eating) to capture the typical events in order to confirm the diagnosis. □

## Legend for video sequence

The seizure occurs midway through dinner while the patient was mixing his food with his right hand and having a conversation. It begins with cessation of mixing of food, blank staring, continual chewing movements, and repeated touching of his head with his left hand. Forty seconds after the clinical onset, horizontal nystagmus and subtle side-to-side head shaking is visible. This is followed by clonic head version to the right, transient generalised stiffening, before the event aborting with a cough. The EEG seizure onset is masked by muscle and chewing artefact. Thirty-two seconds from the clinical onset, an evolving rhythmic 16-Hz activity with admixed sharp components becomes visible bilaterally with a left-sided emphasis. This ictal rhythm gradually evolves into a slower frequency in the theta range with an increasing amplitude. During the last 10 seconds of the seizure, repetitive 4-Hz sharp-wave discharges are seen over the left posterior quadrant. The entire clinical event lasts 2 minutes and 25 seconds.

**Key words for video research on www.epilepticdisorders.com**

*Phenomenology:* focal impaired awareness (complex partial) seizures
*Localisation:* temporal lobe (left)
*Syndrome:* reflex epilepsy (eating epilepsy)
*Aetiology:* idiopathic

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## TEST YOURSELF

(1) What is the typical age at onset for eating epilepsy?

(2) What seizure type is most frequently encountered in eating epilepsy?

(3) During which stage of a meal do patients most often experience seizures?

*Note: Reading the manuscript provides an answer to all questions. Correct answers may be accessed on the website, www.epilepticdisorders.com, under the section "The EpiCentre".*