Eating Epilepsy: An Under-Recognized Form of Reflex Epilepsy

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Abstract
Eating epilepsy is one of the rare forms of reflex epilepsy with less than a hundred cases reported worldwide. The exact pathogenesis still remains unknown and most cases reported in literature are drug resistant. We report the first Algerian case of eating epilepsy presenting with partial complex seizures. Clinical examination was normal. Interictal EEG recording showed asymmetric background activity of lower amplitude and isolated epileptiform activity over the right frontotemporal region. Cerebral MRI revealed right opercular atrophy and treatment with Carbamazepine was successful. Eating epilepsy is often an under-recognized entity whereas its diagnosis is relatively easy. The prognosis varies from one individual to another, but early treatment can improve the quality of life of these patients.

Keywords: Reflex Epilepsy; Eating Epilepsy; Algeria

Introduction
Reflex epilepsy is defined as recurrent seizures exclusively precipitated by specific stimuli [1,2]. Eating epilepsy is one of the rare forms of reflex epilepsy. Indeed, patients with seizures exclusively or predominantly triggered by eating account for 0.5–1/1,000 of all epileptic patients [3], except in Sri Lanka, where it is as high as 148 per 1000 epilepsies [4–6]. Given the important nutritional and psychological complications this entity may generate, its recognition seems to be of great practical interest. Here, we report the first Algerian case of eating epilepsy presenting with complex partial seizures that were associated with feeding.

Observation
The patient was a three year old right-handed girl who was referred to our institute for an evaluation of her seizures. She was born at 41 weeks gestational age via spontaneous vaginal delivery to unrelated healthy parents. No epilepsy or other nervous system diseases were detected in the family history. However, at birth, she exhibited a profile consistent with asphyxia and, subsequently, mild psychomotor developmental delay.

At 30 months of age, patient began to experience complex partial seizures during meals. Ictal semiology was characterised by tonic seizures involving left side of body without loss of consciousness, sometimes associated with adipsic movements of the eyes and head, which could last about five minutes. During these episodes, the child had an empty look and could not answer her parents. Seizures occurred about three times a week and each episode lasted a maximum of thirty seconds. These seizures were triggered by taking the food to the mouth and swallowing at the beginning of the meal, and were mostly provoked by semi-solid meals or consumption of milk.

At baseline clinical examination, no abnormal symptoms or signs were found. Interictal EEG recording showed asymmetric background activity of lower amplitude and isolated epileptiform activity over the right frontotemporal region.

Brain Magnetic Resonance Imaging (MRI) performed using a 1.5-T unit, multiplanar T1 and T2-weighted images; Fluid Attenuation Inversion Recovery (FLAIR) sequences and inversion recovery sequences revealed right opercular atrophy.

Treatment with Carbamazepine 10 mg/kg twice a day was successful. During a three-year follow-up, the patient was in remission and the medication was slowly withdrawn without any seizure relapse.

Discussion
Several factors such as movement, reading, visual stimuli, bath, sleep deprivation, antidepressants, strong emotion and stress have been recognized to trigger epilepsy [7–9]. Eating-induced seizures are paroxysmal manifestations of epileptic nature, triggered exclusively or mostly by meals [3]. Eating epilepsy is considered a rare condition except in Sri Lanka, where several epidemiological studies reported a strikingly high prevalence of eating epilepsy, with familial clusters [5,10]. However, in these cases, the role of specific eating habits such as bulky meals rich in carbohydrates cannot be excluded. To our knowledge, no cases have been reported in Algeria to date.

Our patient began to experience eating-induced seizures at 30 months of age. According to data from the literature, age at seizure onset varies between eight and fifty-five years [11,12] but authentical cases of eating epilepsy have been described in newborns [13]. Seizures occur predominantly at the onset of meals when patients begin to masticate food. Otherwise, these seizures can also occur during breakfast, lunchtime, or after a short period of hunger [14].

Our patient was suffering from complex partial seizures, which represent the most common seizure semiology in eating epilepsy. These seizures are often associated with symptomatic epilepsies, mainly cortical malformation, hypoxic cerebral lesions, primary neuroectodermal tumour and anterior meningoencephalitis [12,15]. However, generalized tonic-clonic seizures, tonic spasms, and status epilepticus have also been reported [16,17].

In our patient, seizures were well controlled with Carbamazepine. Most authors report drug resistance despite polytherapy, justifying surgical intervention or vagus nerve stimulation [18]. In these cases, ketogenic diet which consists of high-fat, low-carbohydrate, and normal-protein diet can be considered as an option. Otherwise, few cases of eating epilepsy responding to conventional anticonvulsants have been reported [12].

The exact pathogenesis of eating epilepsy still remains unknown. Various mechanisms related to eating such as gastric distension [19], mastication and swallowing [20] and chemical composition of food [21] have been proposed as possible stimuli. Several regions of the brain have been implicated in the literature to being involved in initiating eating-induced seizures. Thus, the amygdala seems to play an important role as an integrative centre, particularly in tempo-mesial focal seizures; in other patients, the activation of a perirolandic focus is also possible [3].
In conclusion, we report the first case of eating epilepsy in Algeria. Eating epilepsy is often an under-recognized entity whereas its diagnosis is relatively easy. The prognosis varies from one individual to another, but early treatment can improve the quality of life of these patients.

Declaration of Conflicting Interests

The authors declared no conflicts of interest with respect to the research, authorship, and/or publication of this article.

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