Unilateral Opercular Lesion and Eating-Induced Epileptic Spasms

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Introduction

•**Reflex epilepsy is characterized by seizures precipitated by an identifiable factor or external stimulus.**

•**They are classified into two types: simple and complex. Simple reflex epilepsy is precipitated by simple sensory stimuli such as flashes of light or startle Complex reflex epilepsy is precipitated by complex or more elaborate stimuli such as specific pieces of music or eating.**

•**Although the seizures seen in patients with reflex epilepsy may be of partial or generalized onset, seizures in relation to meals are almost exclusively related to symptomatic focal epilepsy (1).**

•**We describe the imaging and video-EEG data of a patient with a history of treated PNET who developed eating-induced seizures.**

Case Report

Our patient is a 23 year old woman with history of a past left opercular PNET and subsequent right sided weakness after tumor resection and radiation treatment of the tumor at two and a half years of age.

The patient did well until approximately eight years of age when she began to lose control of her head. Without warning, she would suddenly be unable to sustain the upright position of her head, causing it to fall forward. The episodes of head dropping became apparent only during meals during adolescence and at the age of 23, they began occurring exclusively with every meal. At times the seizures would occur multiple times within a single meal, with each seizure lasting less than 5 seconds in duration.

Recent MRI showed left frontal opercular gliosis and volume loss. Video electroencephalography (EEG) monitoring revealed interictal left temporal slowing and frequent left anterior temporal sharp waves. Ictal EEG revealed high voltage (90 to 110 uV) broadly-distributed, frontally-predominant delta slowing, with seizures occurring only during the act of eating.

The patients seizures reduced significantly when her antiepiletpic regimen was changed to valproate (VPA) monotherapy.

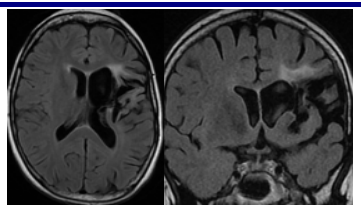

Figure 1. Axial and coronal FLAIR MRI

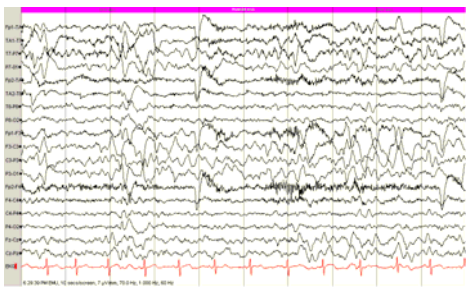

Figure 2. Interictal EEG

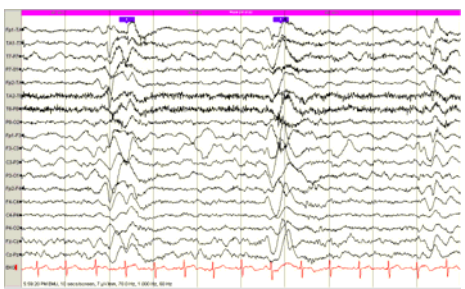

Figure 3. Ictal EEG. Seizures associated with diffuse delta

Discussion

- **The mechanisms of reflex eating epilepsy are poorly understood. Multiple theories have been proposed over the years.**
- **Wieser's critical mass theory proposes that complex reflex epilepsies are in response to a stimulus that triggers a "critical mass" of cortex by recruiting increased amounts of epileptogenic neurons (2).**
- **In eating epilepsy, proposed triggering mechanisms include mastication (3), esophageal stimulation (4), and the satisfactory feeling associated with eating (5).**
- **Multiple studies of animal models with acquired lesions (irritative cortical lesions were created) have been evaluated. In 1929, animal models had seizures induced by photic stimulation after strychnine was applied to the visual cortex. This technique induced seizures of the auditory, gustatory, and olfactory cortex. Lesions made in the visual cortex of rabbits caused regional epileptogenic activity which spread to the masticatory areas, causing seizures to be induced by chewing movements. The EEG spread was representative of cortico-cortical conduction (6).**
- **Other investigators suggest the interaction between temporolimbic and extratemporal regions as being responsible for eating epilepsy (7).**
  - **Hyperexcitability of the temporolimbic area involves susceptibility to gustatory, olfactory, affective, and emotional stimuli. It has been suggested that patients with temporolimbic seizures have constant activation by eating (8). These patients show reflex eating epilepsy from onset, and continue to have most of the seizures with meals.**
  - **Extralimbic (suprasylvian) regions have been implicated when the abnormal cortex is in a proprioceptive region and involves other sensory afferents (lingual, buccal, pharyngeal). These areas are activated by extensive sensory input generated by the complex behaviors involved in eating (8).**
- **Indeed, our patient and others cited in the literature (9, 10, 11) have lesions involving the frontal operculum. Our case appears unique in that the lesion was unilateral, and caused by the patient's prior tumor and subsequent treatment. Although our patient illustrates the fact that the underlying etiology of eating induced seizures is not homogeneous, almost all the reported patients have several elements in common.**
- **Unlike the situation with most of the other reflex epilepsies, the patients with eating-induced seizures uniformly have significant brain injury as an etiology, usually have partial seizures, and are incompletely controlled with medication.**

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