







Case Report 79

Descending Cephalic to Epigastric Sensation in a Patient with Mesial Temporal Lobe Epilepsy: A **Novel Observation**

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Abstract

Keywords

- epilepsy
- ► epigastric aura
- ► mesial temporal lobe epilepsy

One of the most frequent type of auras in mesial temporal lobe epilepsy (MTLE) is epigastric sensation. Until now the site of the symptomatogenic zone of the epigastric aura remains controversial. The temporal lobe as well as insular cortex has been implicated. Our case is that of a 29-year-old young woman patient who presented with an aura of descending cephalic to epigastric sensation as opposed to the prototypical ascending aura. Interictal and ictal recording favored a mesial temporal pattern. Magnetic resonance imaging brain showed left mesial temporal lobe sclerosis. Interictal positron emission tomography showed concordant findings. The patient underwent selective amygdalohippocampectomy following which she remains seizurefree. This previously unreported clinical expression of MTLE and its origins is discussed.

Introduction

Epigastric aura also known as abdominal aura is used to denote a type of somatosensory or somesthetic aura. Epigastric aura includes viscerosensitive sensations such as abdominal discomfort, visceromotor symptoms in the form of tachycardia, vomiting and vegetative symptoms such as

blushing and sweating. Epigastric, gustatory, and olfactory auras are significantly more frequent in patients with hippocampal sclerosis than with other temporal or extratemporal lesions. The sensations are either restricted to the epigastrium/abdomen or are felt as an ascending feeling in the midline thoracic region even up to the pharynx. The

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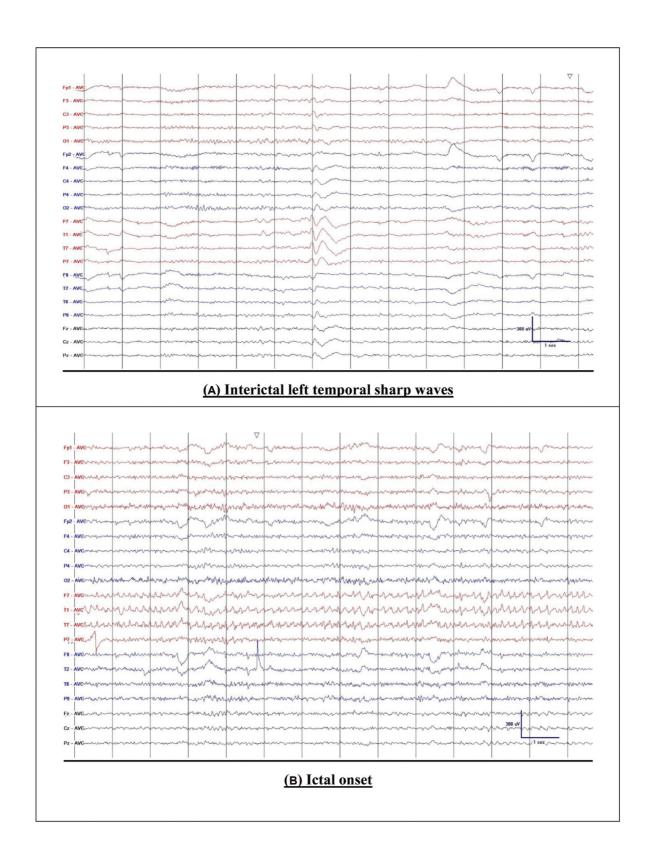
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20

clinical history and video-electroencephalography features of our patient who presented with a descending sensation from the forehead to the epigastrium are described in this article.

Case Report

A 29-year-old young woman patient presented in the epilepsy clinic for recurrent seizures since the age of 22 years. These



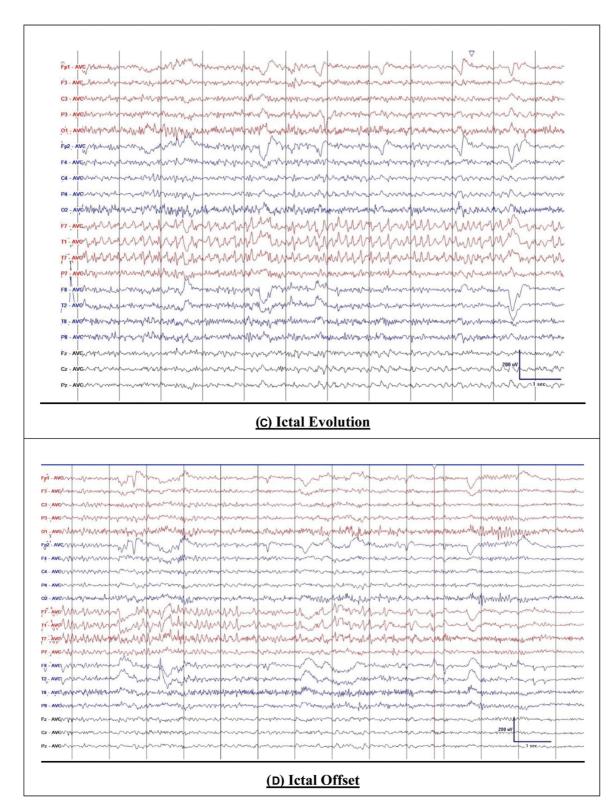


Fig. 1 Common referential average electroencephalography recording with (A) interictal left anterior temporal sharp waves, (B) ictal onset, and (C) evolution of a left anterior and midtemporal rhythmic theta (Ebersole type I rhythm) followed by (D) ictal offset and postictal slowing.

events were characterized by abrupt onset of numbness and a sensation of discomfort beginning from the crown of her head right up to the epigastrium. This may or may not be accompanied by a brief period of impaired awareness. She had no history of a limb or orofacial automatism. Frequency of the events ranged from 3 to 6 per month. The patient was on a low dose of

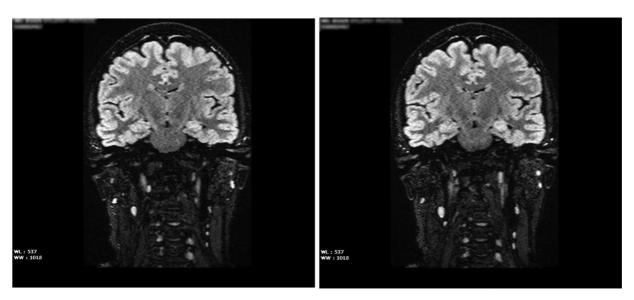


Fig. 2 Magnetic resonance imaging brain—coronal fluid attenuated inversion recovery sequences show hyperintensity in the left hippocampal head and body with loss of digitations and mild volume loss.

carbamazepine (300mg/day). She had no remarkable history of perinatal or childhood neurological insult. She also did not report any history of febrile seizure or family history of seizures. General and neurological examination was unremarkable. Epilepsy monitoring of 48 hours was undertaken during which time the patient had three habitual clinical events. The interictal record (**Fig. 1A**) showed left anterior and midtemporal spike and wave discharges with phase reversals at T1 and positive polarity in C4, P4 (Ebersole type 1 spike).

During the events, the patient had an aura of descending sensation (head to epigastrium) followed by a hypomotor phase (behavioral arrest and impaired awareness lasting 20–30 seconds). Electrographic onset was characterized by rhythmic 8 to 9 Hz activity over the left anterior + midtemporal region. Subsequently, the rhythm turned into a sustained sharply contoured theta in the range 5 to 6 Hz over the same region (**Fig. 1A–D**). Magnetic resonance imaging brain epi-

lepsy protocol revealed a left mesial temporal lobe sclerosis (**Fig. 2**). Antiseizure medication was optimized (carbamazepine 500 mg/day and Clobazam 15 mg/day) following which the patient had no further events for 1 month period. She continued to have repeated episodes of aura (5–10/week) after 1 month despite increasing antiseizure medications. In view of refractoriness, she was given the option of epilepsy surgery. She underwent an interictal positron emission tomography that showed unequivocal hypometabolism of the mesial temporal lobe on the left (**Fig. 3**). She underwent a selective amygdalohippocampectomy following which she is seizure free for the past 3 months.

Discussion

The classical aura of mesial temporal lobe epilepsy is a raising epigastric sensation with or without associated fear or nausea.

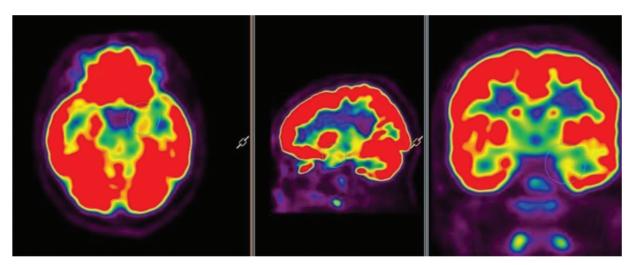


Fig. 3 Interictal fluorodeoxyglucose positron emission tomography—axial, sagittal, and coronal images showing asymmetrical hypometabolism in the left mesial temporal region (circled region).

Some patients may experience abdominal discomfort because of peristaltic contractions. These are all considered to be visceral sensory/motor/autonomic phenomena arising from the limbic system (hippocampus, amygdala, and anterior insula).

In a study by Wang et al of 37 patients with insuloopercular seizures explored by stereo electroencephalography, the clinical and electrographic attributes were categorized into four semiologic subgroups through cluster analysis. The authors found that epigastric sensation was uniformly associated with activation of anteroventral insular regions and the mesial temporal lobe.^{2,3} The insula is also a well-known substrate for somatosensory aura. Based on both animal and human studies, insula is now considered to be the integrating center for viscerosomatosensory function. Insular epilepsy can produce all manner of sensory experiences, the distribution of which can be diffuse, patchy, bilateral, ipsilateral, unilateral, or even midline. Even in patients with typical features of mesial temporal lobe epilepsy, insular contribution can be there. The midline sensory experience of our patient can be a viscerosomatosensory phenomenon projected to the midline region because of insular-mesial temporal lobe activation by the ictus. However, this patient

had typical clinical, electrographic, and radiological features of a mesial temporal lobe epilepsy with a restricted network. Further, the fact that the patient remained seizure free following amygdalohippocampectomy provides substantial evidence of mesial temporal sclerosis being the seat of origin of the descending cephalic to epigastric sensation.

Conflict of Interest None declared.

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