Case Report
Volume 10:06, 2024

Epilepsy Journal

ISSN: 2472-0895 **Open Access**

Involvement of Claustrum in Hippocampal Seizures: A Case Presentation

Rene Andrade Machado*

Division of Pediatric Neurology, Medical College of Wisconsin, Milwaukee, WI, USA

Abstract

The role of claustrum in epilepsy has been documented in animal models. However, no studies of its electrographic activity in humans during hippocampal epileptic seizures exist. The purpose of the present short communication is to show the interictal, ictal, and postictal intracranial electrographic recording of the claustrum in a patient with right temporal lobe epilepsy. A 15-year-old patient with refractory focal temporal lobe epilepsy is presented. Intracranial recording and wavelet transform analysis show a particular background and ictal pattern of claustrum during a hippocampal seizure. In conclusion, this report suggests the possible involvement of the claustrum in temporal lobe epileptic seizures in humans.

Keywords: Claustrum • Seizures • Intracranial recording • High frequency oscillations • Temporal lobe epilepsy

Introduction

The claustrum is a thin mass of gray matter located between the insular cortex and the external capsule [1]. Its role in epilepsy has been documented in animal models [2]. However, no studies of its electrographic activity in humans during hippocampal epileptic seizures exist. The purpose of the present correspondence is to show the interictal, ictal, and postictal intracranial electrographic recording of the claustrum in a patient with right temporal lobe epilepsy.

Case Presentation

This is a 15-year-old adolescent patient, male, right-handed, with a history of epilepsy, being treated with Lamotrigine 300 mg per day, Levetiracetam, 4000 mg/day, and clobazam 20 mg daily. He reports seizures consisting of sudden, brief events characterized by an ascending epigastric sensation followed by gestural and bipedal automatisms and postictal confusion. The frequency of epileptic seizures was 2 per week. Postictal amnesia was documented.

A complete presurgical evaluation included video-electroencephalogram; Magnetic Resonance Imaging [MRI], Positron Emission Tomography [PET], and neuropsychological assessment were performed.

The clinical history and presurgical evaluation were presented at the institution's multidisciplinary epilepsy meeting. It was decided to continue to assess the ictal onset zone using intracranial Stereoelectroencephalography (SEEG) recordings. SEEG electrodes were planned to be implanted in the hippocampus, amygdala, right superior, middle, and inferior temporal gyri, insula, and right orbitofrontal cortex (Figure 1A implantation schematic). In our hospital, implanting electrodes in the basal ganglia is not routine. However, in this patient, incidentally, the number one contact of the PI electrode (posterior insular) was placed in the claustral cortex (Figure 1B).

Subsequently, a quantitative analysis of electrocorticographic activity was performed. To this end, the electrocorticography signals were processed using the average reference method to eliminate activity common to all electrodes and improve the signal noise. Subsequently, the discrete wavelet transform, DWT, was applied.

The evaluation of intracranial ictal patterns and the presentation of their results were accepted by the scientific and ethical committee of the institution according to the protocol contained in minutes 00030622, project PRO00049035.

Results

The video of the recorded seizures showed the presence of focal epileptic seizures with impairment of awareness and gestural automatisms. The electroencephalogram showed rhythmic activity at 5.6 Hz in the right temporoparietal region. The MRI showed no structural lesion, and the PET showed an area of right neocortical temporal and orbitofrontal hypometabolism (Figure 1). The neuropsychological evaluation showed a verbal IQ of 95 and a visuospatial IQ of 100, with no discrepancies between the verbal and visuospatial quotients. The electroencephalogram did not show interictal epileptiform activity. The clinical history and presurgical evaluation were presented at the institution's multidisciplinary epilepsy meeting. It was decided to continue to assess the ictal onset zone using intracranial Stereoelectroencephalography (SEEG) recordings.

During the intracranial evaluation, nine right hippocampal epileptic seizures were recorded. A right amygdalo-hippocampectomy was performed, and the patient remained seizure-free during six months of postsurgical follow-up. The biopsy showed right hippocampal sclerosis.

The Discrete Wavelet Transform (DWT) results are shown in Figures 2 and 3.

Discussion

This patient presents frequent seizures despite treatment with three antiseizure medications. Thus, it was established that the patient had criteria to be considered to have drug-resistant epilepsy [3]. The presence of a subjective symptom (rising epigastric sensation) forced the epileptogenic area to be located in a central autonomic integration structure: The amygdala-hippocampal region, the orbitofrontal cortex, the insula, or the cingulum [4].

***Address for Correspondence:** Rene Andrade Machado, Division of Pediatric Neurology, Medical College of Wisconsin, Milwaukee, WI, USA, Tel: 3138044803, E-mail: reneandrade1970@yahoo.es*

Received: 14 November 2024, Manuscript No. elj-24-152518; **Editor assigned:** 16 November 2024, PreQC No. P-152518; **Reviewed:** 28 November, 2024, QC No. Q-152518; **Revised:** 05 December 2024, Manuscript No. R-152518; **Published:** 12 December 2024, DOI: 10.37421/2472-0895.2024.10.286

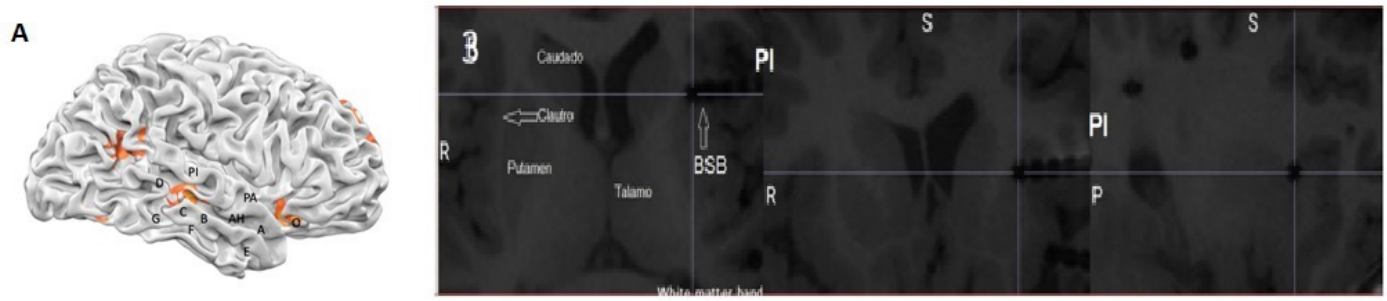

Figure 1. SEEG electrode implantation scheme and their location. Pi means electrode with the target in the insula, posterior region. R indicates the right region of the MRI, BSB means white Matter Band, and O, E, AH, B, G, F, PI, and PA are the nomenclature of the different electrodes. O destined for the orbitofrontal region, PA and PI for the anterior and posterior insula, AH for the hippocampus, and B, C, and D for the body and posterior region of the hippocampus, and parahippocampal region successively. F and G were destined for the inferior temporal gyrus—electrode E to the temporopolar region and amygdala. Areas in orange represent hypometabolic areas in PET.

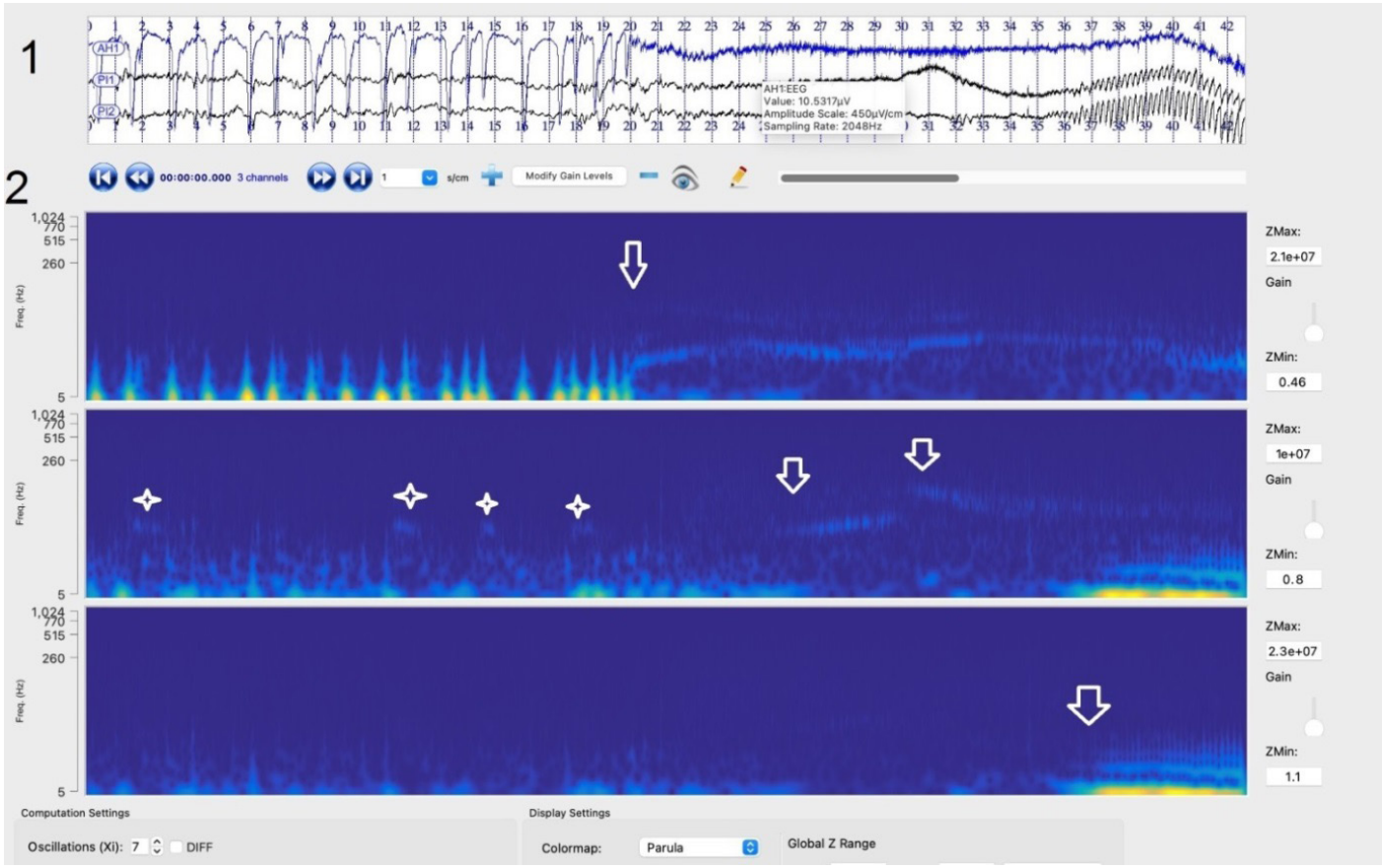

Figure 2. Pericatal electrical activity recorded in the patient seconds before one of the seizures. Three channels are shown in the upper panel: AH1 hippocampus, PI1, claustrum cortex, and PI2 in the insula. Panel 2 shows the wavelet transform. Pericatal activity was characterized by high-amplitude periodic spikes and waves in the hippocampus (Figure 2 upper panel [1]). The lower SEEG channels point to electrodes PI1 and PI2 located in the claustrum and insula respectively. Note the difference in interictal activity with star-marked High-Frequency Oscillations (HFOs) occurring periodically in PI1 (105 Hz) and not in PI2. The background activity in PI1 (claustrum) is fundamentally alpha and beta (8.5-14.5 Hz), while in the insula, it is theta-delta (3-7.5 Hz) observed electrode (PI2) Figure 2 panel 2. Arrows mark the seizure onset in each contact.

The absence of a structural lesion (non-lesional temporal epilepsy), with non-concordant findings between the ictal electroencephalogram and the hypometabolism pattern of the PET, made us consider the possibility of continuing the presurgical evaluation using intracranial recordings. For this reason, a scheme for the implantation of intracranial SEEG electrodes was developed, with the structures shown in Figure 1 as possible targets.

Nine epileptic seizures were recorded, all originating in the right hippocampus. The absence of epileptic seizures after right amygdalo-hippocampectomy confirms that the epileptogenic zone corresponded to the right mesial temporal structures.

The results shown in Figures 2 and 3 show two seizures coming from the right hippocampus, electrode HA1 contact 1 (panel 1 Figures 2 and 3). The wavelet transform shows a different pericatal and ictal electroencephalographic pattern in the PI contact 1 electrode and PI contact 2. This means that they record electrographic activity from different brain structures. MRI shows contact PI1 in the claustrum while PI2 in the insula (Figure 1B). Therefore, we determined that the electrographic activity recorded belongs to the claustrum. The claustrum seems to participate in the epileptogenic network. Background activity in the claustrum ranged between the alpha and beta frequencies. Interictal epileptiform discharges were characterized by periodic High Frequency Oscillation (HFO) synchronous with the hippocampal interictal activity, the same happened during epileptic seizures (Figure 2).

This is the first reported case of periictal, ictal claustral activity during a hippocampal seizure in humans. MRI studies have shown that after epileptic seizures, cytotoxic edema can be observed in the claustrum, which has indirectly suggested that these neurons discharge during epileptic seizures [5-7]. Furthermore, anatomopathological studies have documented neuronal loss

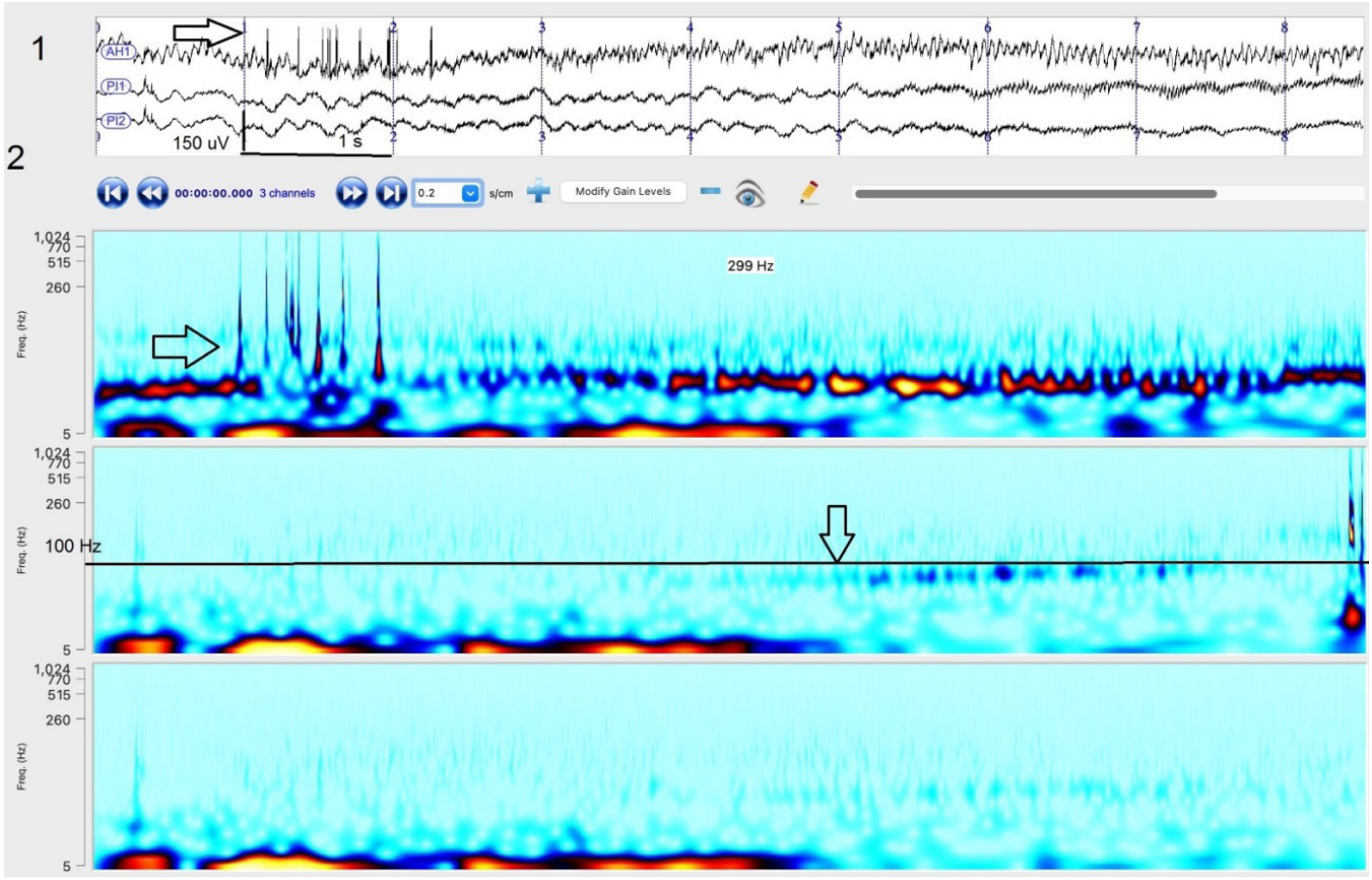

Figure 3. Ictal activity in the claustrum during an epileptic seizure originating in the hippocampus. Epileptic seizure recorded in the hippocampus. The beginning is marked by rhythmic spikes at 5 Hz with HFO >200 Hz and followed by low-amplitude and high-frequency activity (observe wavelet HFO that reaches 260 Hz; 3 seconds later, HFO is evident in the claustrum (PI1) while in Insula electrode (PI2) shows a drop in frequencies below 5 Hz.

in the claustrum, secondary to seizures in laboratory animals [8]. Koubeissi, et al., for their part, showed the role of the claustrum in the loss of consciousness in patients with epileptic seizures [2]. The propagation of ictal electrographic activity towards the right claustrum in our patient could determine the loss of consciousness during his epileptic seizures. To know these connections in detail, I refer the reader to the study by Honda Y, et al. published in "The Journal of Comparative Neurology" in 2024 [9].

Our patient did not have a lesion in the insula; there was no insular hypometabolism in the PET scan, and the seizures were not typical for this location [8], which rules out the insula as an area of ictal onset. Furthermore, intracranial recordings did not show involvement of the insula (see electrode PI2) at least in the first 10 seconds of the epileptic seizure. This suggests that the activity seen in electrode PI contact 1 is in fact a claustrum electrographic activity.

Studies in mice have shown that the claustrum is activated during limbic epileptic seizures [7,8]. These studies further show that the claustrum usually generates and maintains ictal epileptiform discharges. On the other hand, they show that claustrum lesions can prevent the development of kindling in the amygdala in experiments with laboratory animals [8]. These results suggest a fundamental role of the claustrum in epileptic seizures. Thus, neuromodulation of the claustrum could be a future target in treating temporal lobe epilepsy or perhaps in blocking the mechanisms that determine the development of primary or secondary epileptogenesis.

Conclusion

In conclusion, this report suggests the possible involvement of the claustrum in temporal lobe epileptic seizures in humans.

Acknowledgement

None.

Declaration of Interest

None.

References

1. Jackson, Jesse, Jared B. Smith and Albert K. Lee. "The anatomy and physiology of claustrum-cortex interactions." *Annu Rev Neurosci* 43 (2020): 231-247.
2. Pavel, Bogdan, Fabien Menardy, Diana Rotaru and Alexandru Catalin Paslaru, et al. "Electrical stimulation in the claustrum area induces a deepening of isoflurane anesthesia in rat." *Brain Sci* 9 (2019): 304.
3. Hitiris, Nikolas, Rajiv Mohanraj, John Norrie and Graeme J. Sills, et al. "Predictors of pharmacoresistant epilepsy." *Epilepsy Res* 75 (2007): 192-196.
4. Abou-Khalil. "Semiology of temporal lobe epilepsies." *Neurosci* 3(2003): 139-42.
5. Guo, Kundian and Zhen Hong. "Claustrum sign in Febrile Infection-Related Epilepsy Syndrome (FIRES)." *Neurol Sci* 44 (2023): 3357-3359.
6. Meletti, Stefano, Giada Giovannini, Giuseppe d'Orsi and Lisa Toran, et al. "New-onset refractory status epilepticus with claustrum damage: Definition of the clinical and neuroimaging features." *Front Neurol* 8 (2017): 111.
7. Chen, Shaoyun, Masayuki Kobayashi, Yoshiko Honda and Sachiko Kakuta, et al. "Preferential neuron loss in the rat piriform cortex following pilocarpine-induced status epilepticus." *Epilepsy Res* 74 (2007): 1-18.

8. Zhang, Xia, Darren K. Hannesson, Deborah M. Saucier and Amy E. Wallace, et al. "Susceptibility to kindling and neuronal connections of the anterior claustrum." *Neurosci J* 21 (2001): 3674-3687.

9. Honda, Yoshiko, Keiko Moriya-Ito, Tetsuya Shimokawa and Yasushi Kobayashi. "Connectivity of the claustrum–endopiriform complex with the presubiculum and hippocampal regions in the common marmoset (*Callithrix jacchus*)." *J Comp Neurol* 532 (2024): e25666.

How to cite this article: Machado, Rene Andrade. "Involvement of Claustrum in Hippocampal Seizures: A Case Presentation." *Epilepsy J* 10 (2024): 286.