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BRIEF COMMUNICATION

Neuroanatomical localization of faciobrachial dystonic seizures in LGI1-antibody encephalitis

Soo Hyun Ahn[1] | **Maryam Karaminiya**[2] | **Yihui Goh**[1,3,4] | **Woo-Seok Ha**[5] | **Yoonhee Shin**[1] | **Yoonhyuk Jang**[1,6,7] | **Samantha Banks**[8] | **Ki-Young Jung**[1] | **Kon Chu**[1] | **Sang-Kun Lee**[1] | **Kyung Min Kim**[5] | **Seung Bong Hong**[9] | **Sarosh Irani**[2] | **Soon-Tae Lee**[1]

[1]Department of Neurology, Seoul National University Hospital, Seoul National University College of Medicine, Seoul, South Korea

[2]Department of Neurology, Mayo Clinic, Jacksonville, Florida, USA

[3]Division of Neurology, Department of Medicine, National University Health System, Singapore, Singapore

[4]Department of Medicine, Yong Loo Lin School of Medicine, National University of Singapore, Singapore, Singapore

[5]Department of Neurology, Yonsei University College of Medicine, Seoul, South Korea

[6]Biomedical Research Institute, Seoul National University Hospital, Seoul, South Korea

[7]National Strategic Technology Research Institute, Seoul, South Korea

[8]Department of Neurology, Mayo Clinic Rochester, Rochester, Minnesota, USA

[9]Epilepsy Center, Department of Neurology, St. Peter's General Hospital, Seoul, South Korea

Correspondence

Soon-Tae Lee, Department of Neurology, Seoul National University Hospital, Seoul National University College of Medicine, 101 Daehak-ro, Jongno-gu, Seoul 03080, South Korea.
Email: staelee@snu.ac.kr

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Abstract

Faciobrachial dystonic seizures (FBDS), paroxysmal dizziness spells, and thermal sensory attacks are highly frequent and stereotypic phenomena experienced in leucine-rich glioma inactivated 1 (LGI1)-antibody encephalitis. This study aims to describe the electrophysiologic mechanism underlying these pathognomonic symptoms. LGI1-antibody encephalitis patients with active symptoms were enrolled from two separate centers in South Korea and the United States. Patients were evaluated with simultaneous magnetoencephalography (306 channels) and electroencephalography. Regional alterations in neuronal excitability represented by interictal epileptiform discharges were present in the faciobrachial area of the motor cortex, insula, and somatosensory cortex, somatotopically aligned with each of the ictal semiologies observed in patients. FBDS and other LGI1-antibody encephalitis-specific spells localized to cortical regions neuroanatomically corresponding to ictal semiologies: the faciobrachial homunculus (FBDS), insular cortex (paroxysmal dizziness spells), and somatosensory cortex (thermal sensory attacks). Our findings support the ictal hypothesis underlying these unique phenomena.

Soo Hyun Ahn and Maryam Karaminiya contributed equally as co-first authors.

KEYWORDS
faciobrachial dystonic seizure, LGI1-antibody encephalitis, localization, magnetoencephalography, semiology

1 | INTRODUCTION

Leucine-rich glioma inactivated 1-antibody encephalitis (LGI1-Ab-E), the most common form of autoimmune encephalitis, typically presents with faciobrachial dystonic seizures (FBDS), a hallmark feature characterized by brief dystonic jerks of the face and arm.[1,2] Other semiologies include piloerection, thermal paresthesia, and paroxysmal dizziness.[2] These seizures often lack corresponding electroencephalographic (EEG) changes, meaning they are commonly mistaken for functional attacks.[2] Although putative cellular mechanisms underlying LGI1 antibody-mediated epileptogenesis have been reported,[2] the neuroanatomical basis underlying the distinct clinical manifestations of FBDS remains debated.

Magnetoencephalography (MEG) is a highly sensitive tool for detecting cortical neuronal activity, offering high temporal and spatial resolution. Its technical advantages enable detection of neural signals that are often imperceptible on EEG.[3] Here, we present a neuroanatomical explanation for FBDS and its variants through MEG analysis.

2 | MATERIALS AND METHODS

Simultaneous MEG (306 channels) and EEG recordings were acquired from patients with LGI1-Ab-E presenting with active symptoms and frequent FBDS at two centers: Seoul National University Hospital and Mayo Clinic, USA. Magnetic source localization was performed using single or multiple equivalent current dipole (ECD) models. Video monitoring was performed during MEG to confirm FBDS events. Clinical information for all patients and the MEG report of one patient (Mayo Clinic) were obtained by medical chart review. This study was approved by the institutional review boards of both participating institutions.

3 | RESULTS

Seven patients (five male, two female; mean age = 53 ± 13.1 years), all with serum LGI1-IgG, presented with typical clinical features (Table 1) and underwent MEG. All patients had active seizures at the time of testing, including varying degrees of FBDS or related semiologies. MEG revealed interictal epileptiform discharges (IEDs) that were not observed on prior or concurrent EEGs.

ECD modeling localized dipoles to cortical regions with clear neuroanatomical alignment to patient-reported symptoms (Figure 1A,C). Patients with active FBDS exhibited contralateral interictal discharges that appeared in proximity to clinical symptoms, within 1–2 s (ID 1, 7; Figure 1B, Table 1), with dipoles localized either to the primary motor cortex, specifically the faciobrachial area of the motor homunculus, or to the medial temporal lobe. The patient with paroxysmal dizziness spells exhibited IEDs in the insular cortex (ID 2). Patients with recurrent thermal sensory attacks had dipoles localized to the somatosensory cortex (ID 3, 4). Typical electrographic seizures were absent in all patients.

4 | DISCUSSION

The electrophysiological basis and localization of FBDS and associated distinctive semiologies in LGI1-Ab-E have remained elusive. Our findings suggest involvement of distinct neuroanatomical regions corresponding to the specific semiologies of FBDS and other LGI1-Ab-E-associated seizures: the face and arm regions of motor cortex for classical FBDS, the somatosensory cortex for thermal sensory attacks, and the vestibular insular cortex for paroxysmal dizziness spells. These differential localizations to somatotopically relevant cortical areas suggest regionally altered neuronal excitability, likely mediated by LGI1 antibodies.[2]

Previous MEG studies utilizing jerk-locked back averaging have demonstrated involvement of the premotor cortex in FBDS.[4] Detailed EEG analyses have also suggested a role for primary motor cortex in FBDS, in conjunction with the striatum.[5] LG11-Ab-E is frequently associated with T1 signal changes in the basal ganglia, and functional imaging studies have reported hypermetabolism in the basal ganglia and thalamus.[6,7] Taken together, these findings support the presence of a complex epileptic network in LGI1-Ab-E encompassing both cortical and subcortical structures. Accordingly, the variable cortical involvement observed in our study, which may contribute to distinct clinical semiologies, should be interpreted within this network-level framework.

In addition to localization, these findings further support the epileptic nature of FBDS and other short-lived

TABLE 1 Patient clinical information.

ID	Age, years; sex	Symptoms	FBDS and its variants semiology	MRI FLAIR change	EEG findings	MEG findings
1	42; F	FBDS, memory impairment, psychiatric symptoms	Brief dystonic jerks in both arms, with stinging pain in right shoulder, occipital area, and leg	Both medial temporal lobes	NA	Spikes in right mid-posterior–medial–basal temporal regions, including hippocampus
2	61; M	FBDS, paroxysmal dizziness spells, psychiatric symptoms, memory impairment, focal to bilateral tonic–clonic seizures	Dystonic jerks in both arms (right > left)	Normal	Small amount of intermittent generalized theta slow waves	Spikes and sharp transients in the right midparietal, left mid-parietal, and insular regions
3	56; M	FBDS, thermal paresthesia in left arm and leg, memory impairment	Brief jerks in right arm and leg Sharp electric pain in left arm, spreading to occipital area, bilateral arms and legs	Left medial temporal lobe	Normal EEG	Sharp transients in the right temporoparieto-occipital junction, left postcentral gyrus, and medial occipital region
4	48; M	FBDS, memory impairment	Dystonic jerk in right arm and leg, followed by numb pain in right arm, face, and leg	Normal	Fast activity in the central area	Sharp transients in the right insular, parietal, left midtemporal, posterior–superior temporal, and insular regions
5	31; F	FBDS, recurrent nausea and piloerection in the right arm, déjà vu, memory impairment, homonymous hemianopia	Recurrent chills in the right arm and hand, with or without brief dystonic jerks	Both medial temporal lobes	Few interictal discharges in the left temporal area	Spikes and sharp transients in the right occipital lobe
6	73; M	FBDS, memory impairment, confusion, falls	Brief dystonic jerks in both arms and face (left > right)	Right medial temporal lobe	Intermittent generalized theta slowing	Sharp transient in the right occipital region
7	63; M	FBDS, paroxysmal dizziness spells, imbalance, short-term memory impairment, hyponatremia	Brief jerks in left arm, spreading to right arm, face, and leg with preserved awareness	Right hippocampus (atrophy)	Normal EEG	Discharges that were not definitively epileptiform just anterior to the right precentral gyrus within 1–2 s to FBDS onset

Note: Patient ID 1–6 were enrolled from Seoul National University Hospital. Patient ID 7 was enrolled from Mayo Clinic.

Abbreviations: EEG = electroencephalogram; F = female; FBDS = faciobrachial dystonic seizures; FLAIR = fluid-attenuated inversion recovery; M = male; MEG = magnetoencephalographic; MRI = magnetic resonance imaging; NA = not available.

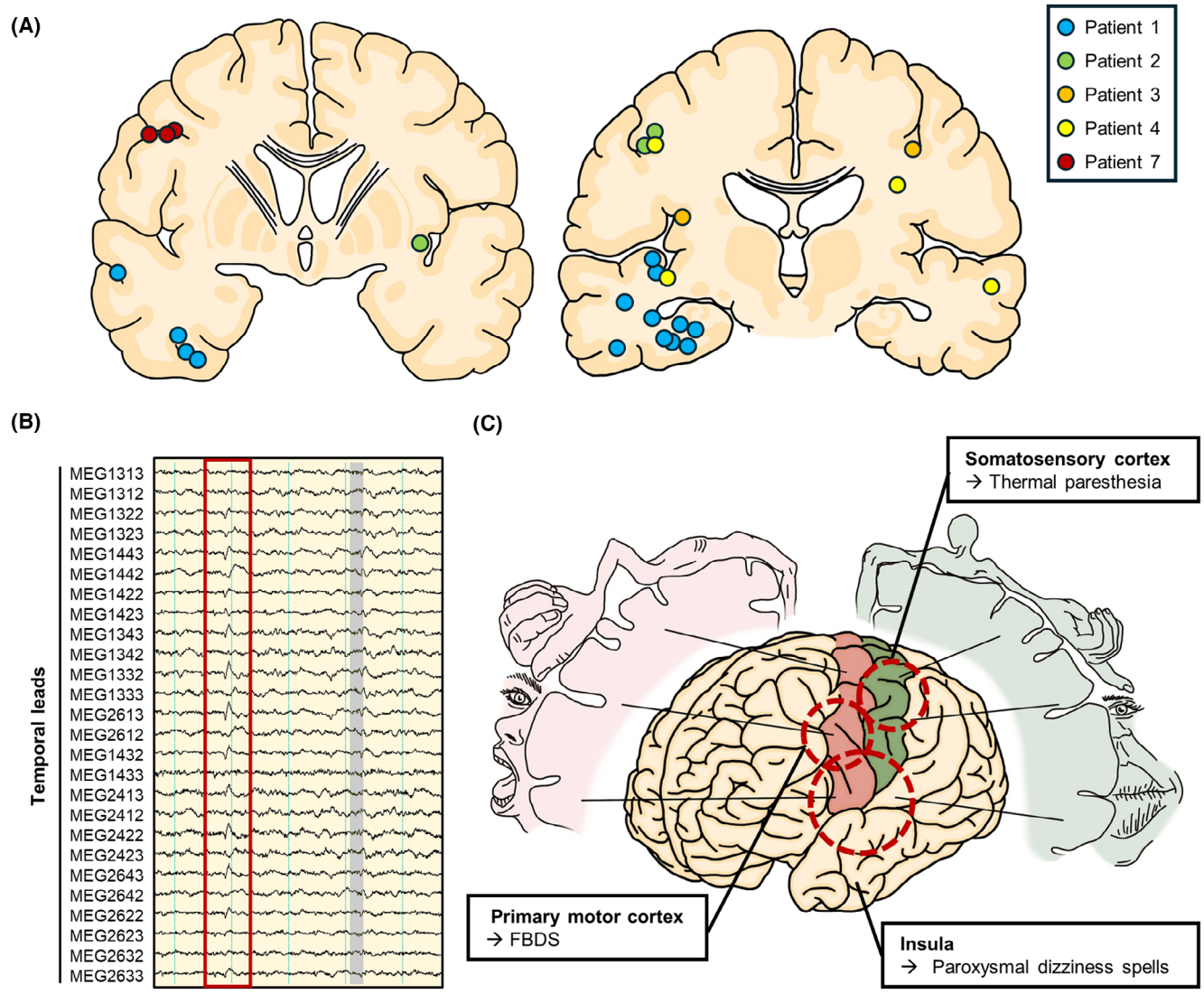

FIGURE 1 Magnetoencephalography (MEG)-based cortical source localization of epileptiform discharges in LGI1 (leucine-rich glioma inactivated 1) encephalitis. (A) Composite coronal sections showing group-level localization of IEDs. IEDs from individual patients are color-coded. Two patients (ID 5, 6) had occipital discharges that are not depicted in this figure. (B) A representative spike (highlighted in red) localized to the medial temporal region shortly preceding a faciobrachial dystonic seizure (FBDS; gray box) suggests hyperexcitability and possible activation of epileptic network in the cortical area contralateral to patient symptoms. (C) Schematic illustration of neuroanatomical correspondence between MEG-derived IEDs and recurrent clinical symptoms.

phenomena in patients with LGI1-Ab-E. Although typical ictal recordings were not captured, IEDs represent synchronous neuronal firing within local cortical areas that likely constitute components of the epileptic network.[8] This phenomenon is likely mediated by LGI1 antibodies, which are capable of inducing neuronal molecular disruptions and hyperexcitability.[2] Antibody-mediated ictogenesis and subsequent epileptogenesis can induce acute symptomatic seizure or focal autoimmune-associated epilepsy.[9]

Limitations may include the relatively small number of cases and potential insensitivity of MEG. Nevertheless, our study is the largest MEG series in LGI1-Ab-E to date and overcame a major challenge of recruiting actively unwell, seizing patients. Further studies incorporating network analysis, event-related field mapping, and higher resolution electrophysiologic techniques such as electromyographic/MEG polygraphy that allow accurate symptom-event correlation may provide more information about the neuroanatomical localization of FBDS semiology.

AUTHOR CONTRIBUTIONS

Soo Hyun Ahn, Sarosh Irani, and Soon-Tae Lee contributed to the conception and design of the article. All authors contributed to the acquisition, analysis, and interpretation of data. Soo Hyun Ahn, Maryam Karaminiya, Sarosh Irani, and Soon-Tae Lee contributed to drafting the

text and preparation of figures. All authors contributed to critical review of the manuscript. Sarosh Irani and Soon-Tae Lee supervised this study.

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CONFLICT OF INTEREST STATEMENT

S-T.L. serves on advisory boards for Roche/Genentech, Arialys, Argenx, Advanced Neural Technologies, and Piehealthcare. S.I. has received honoraria/research support from Amgen, Argenx, UCB, Roche, Janssen, IQVIA, Clarivate, Slingshot Insights, Cerebral Therapeutics, BioHaven Therapeutics, CSL Behring, and ONO Pharma; receives licensed royalties on patent application WO/2010/046716 entitled "Neurological Autoimmune Disorders"; and has filed two other patents entitled "Diagnostic Method and Therapy" (WO2019211633 and US app 17/051930; PCT application WO202189788A1) and "Biomarkers" (WO202189788A1, US App 18/279624; PCT/GB2022/050614). The remaining authors have no conflicts of interest. We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ORCID

Sang-Kun Lee https://orcid.org/0000-0003-1908-0699
Soon-Tae Lee https://orcid.org/0000-0003-4767-7564

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