Suspected LGI1 Autoimmune Encephalitis With Basal Ganglia and Insular MRI Abnormalities: A Case Report

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Received: 8 October 2025 · Published: 31 October 2025

Abstract

A 73-year-old man presented after recurrent involuntary movements involving the arm and face, followed by an episode concerning for seizure. Hyponatremia and episodic movements raised suspicion for an autoimmune process. Brain MRI showed evolving left basal ganglia abnormalities and a new left insular signal abnormality. The differential diagnosis included ischemia and autoimmune encephalitis. The treating team favored LGI1 autoimmune encephalitis and administered high-dose corticosteroids and intravenous immunoglobulin. The report describes clinical improvement and resolution of the illustrated MRI abnormalities after treatment. The case illustrates the role of clinical chronology and serial imaging when focal MRI abnormalities initially raise concern for stroke.

Keywords: LGI1 encephalitis, autoimmune encephalitis, basal ganglia, magnetic resonance imaging, seizures

Introduction

LGI1 autoimmune encephalitis can present with faciobrachial dystonic seizures, cognitive or behavioral changes and hyponatremia. Focal imaging abnormalities can overlap with other diagnoses. This case illustrates the interpretation of evolving basal ganglia and insular MRI findings in the context of the clinical course [1,2,3].

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ISSN 2813-7221 · Swiss J. Radiol. Nucl. Med. 2025 24(1):8-14 · DOI 10.59667/sjoranm.v24i1.16

Clinical Case

A 73-year-old man with hypothyroidism and lumbar stenosis presented with agonal breathing and concern for an unwitnessed seizure. His wife reported intermittent involuntary contractions of the left arm and face over approximately two weeks, with preserved awareness, occurring about three times per hour or more. She also described a later episode of right arm contraction and shaking with tongue biting and bleeding, without observed eye deviation. The earlier episodes preceded outpatient methylprednisolone and gabapentin.

At an earlier evaluation for left arm jerking, the report describes bilateral upper limb tremor with an otherwise normal motor and sensory examination. Sodium was 127 as reported. Glucose and creatinine were reported as 107 and 0.9, respectively. CBC was unremarkable and ECG showed sinus rhythm. Cervical CT showed no acute abnormality. Tremor resolved after morphine and orphenadrine at that visit, and baclofen and naproxen were prescribed on discharge.

Investigations

Levetiracetam was continued for recurrent movements. EEG showed widespread slowing and disorganization with rare intermittent left temporal slowing. Occasional synchronous single jerks of the left arm and leg were observed. The report describes no explanatory acute finding on CT or CT angiography. MRI showed evolving left basal ganglia abnormalities, with ischemia and autoimmune encephalitis considered, and a new left insular signal abnormality (Figure 1). Diffusion restriction was described in the illustrated images.

CSF results were reported as WBC 2, RBC 7, glucose 75, protein 46, IgG index 0.72 and IgG synthesis rate 3.4.

Treatment and Reported Course

The treating team favored LGI1 autoimmune encephalitis and treated the patient with high-dose corticosteroids and intravenous immunoglobulin. The report describes resolution of symptoms and improvement of the illustrated MRI abnormalities (Figure 2). Oral prednisolone was started and outpatient neuroimmunology follow-up was recommended.

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ISSN 2813-7221 · Swiss J. Radiol. Nucl. Med. 2025 24(1):8-14 · DOI 10.59667/sjoranm.v24i1.16

MRI Findings Before Treatment

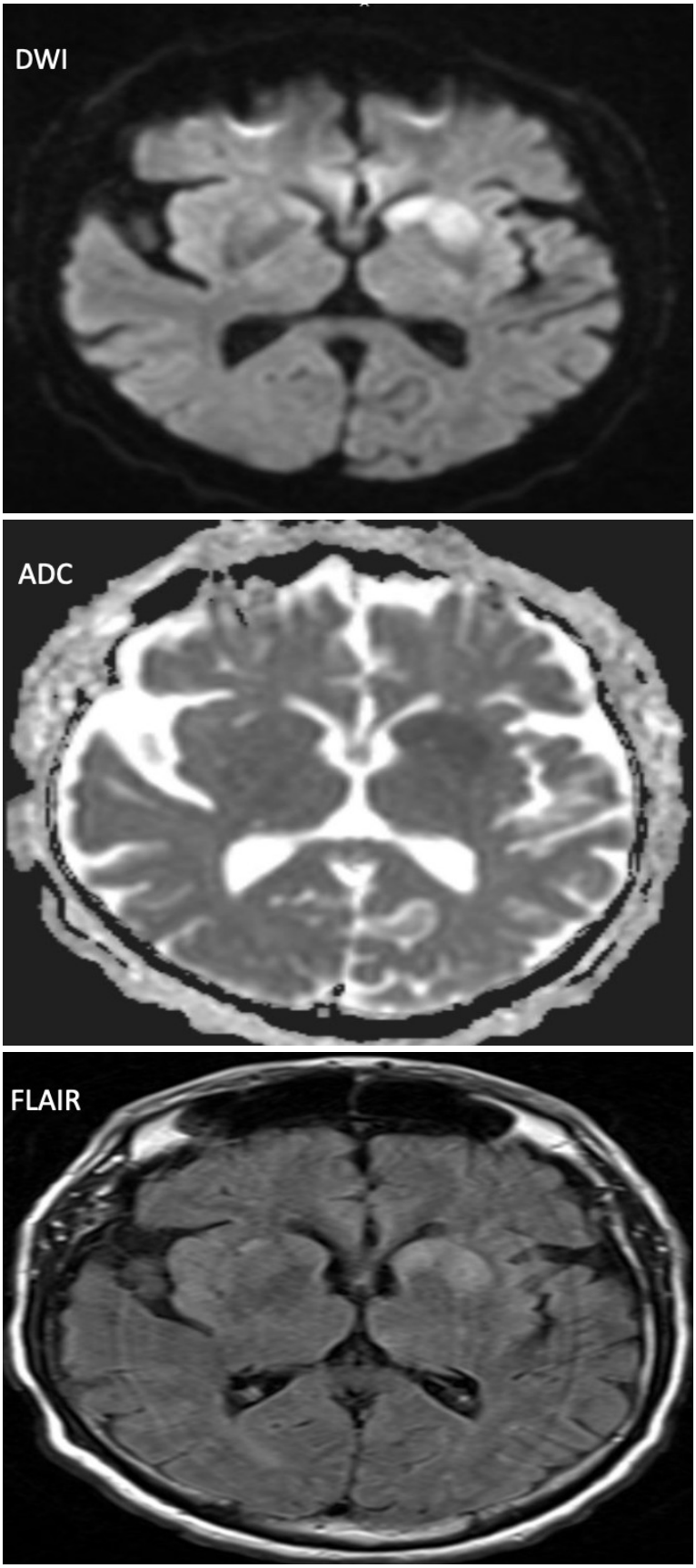


Figure 1. Evolving left basal ganglia findings and new left insular signal abnormality. The initial imaging differential included ischemia and autoimmune encephalitis. High diffusion-weighted signal with low ADC was described.

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MRI Findings After Treatment

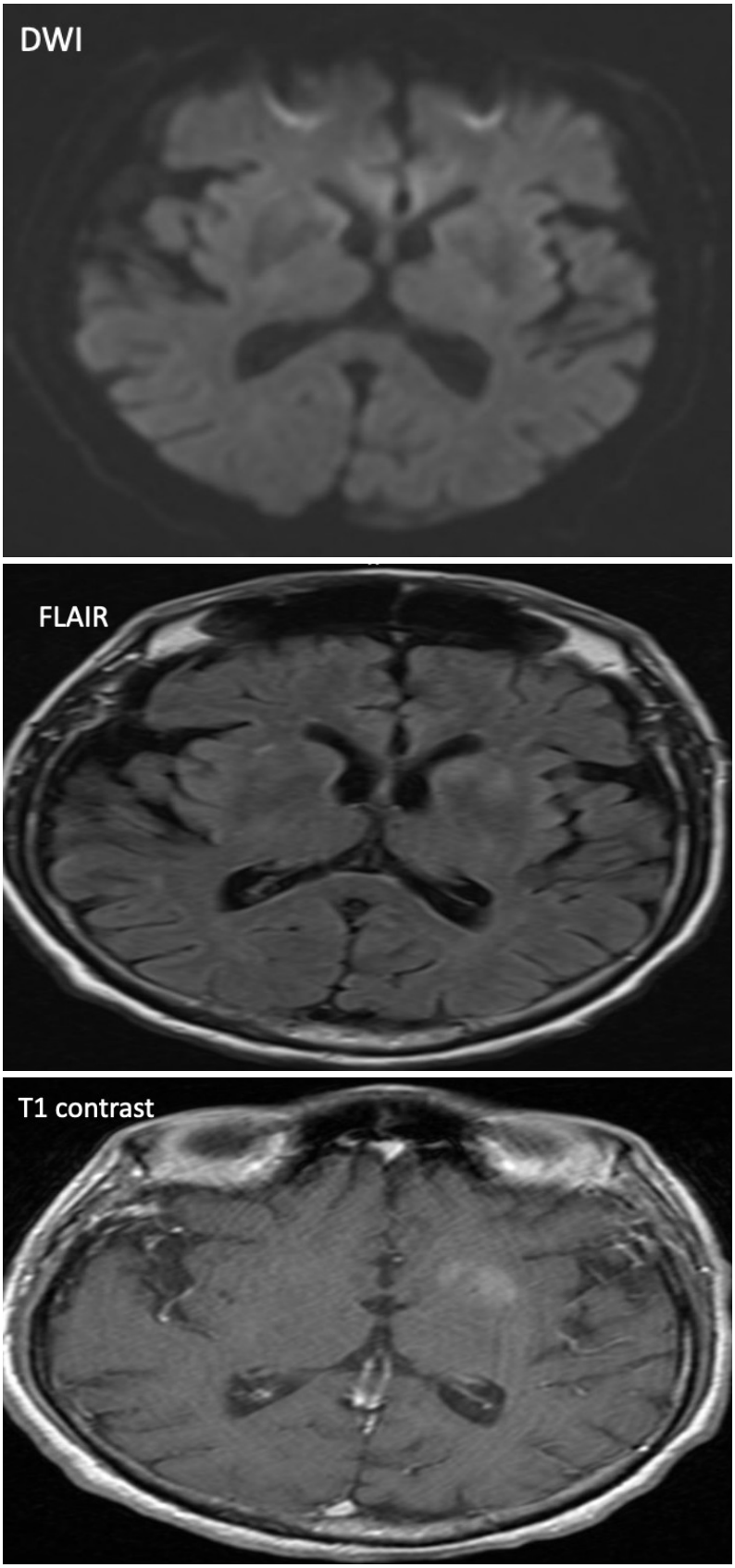


Figure 2. Follow-up MRI after corticosteroids and intravenous immunoglobulin. The report describes resolution of the illustrated abnormalities.

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Discussion

The episodic arm and facial movements, hyponatremia and reported cognitive symptoms supported consideration of an autoimmune process. LGI1 participates in synaptic signaling through interactions with ADAM22 and ADAM23. Clinical manifestations and imaging findings vary [1,2,3,4,5,6,7,8,9,10,11,12,13,14].

Basal ganglia or temporal signal abnormalities may occur in this setting, but imaging patterns can overlap with other etiologies. Clinical chronology, EEG, serial imaging and available laboratory findings should be assessed together [15,16,17,18,19,20,21,22,23]. The working diagnosis in this case was suspected LGI1 autoimmune encephalitis. The observed response to immunotherapy does not establish the etiology by itself.

Corticosteroids, intravenous immunoglobulin and plasma exchange are discussed in the literature on autoimmune encephalitis. Treatment decisions and follow-up depend on the clinical context [24,25,26,27,28]. In this patient the report documents corticosteroids and intravenous immunoglobulin, followed by prednisolone and a recommendation for specialist follow-up.

Conclusion

In this case, the clinical chronology and evolving MRI findings led the treating team to consider autoimmune encephalitis in the differential diagnosis of focal basal ganglia abnormalities. The reported clinical and imaging improvement after immunotherapy supports the clinical assessment, while the cause remains a working diagnosis.

Declarations

The authors report written informed consent for publication, preservation of patient anonymity and compliance with the Declaration of Helsinki and applicable standards. No competing interests or funding were reported.

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ISSN 2813-7221 · Swiss J. Radiol. Nucl. Med. 2025 24(1):8-14 · DOI 10.59667/sjoranm.v24i1.16

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