CASE REPORT

Autoimmune encephalitis with coexistent LGI1 and GABA<sub>B</sub>R1 antibodies: case report

Yi Xie††, Jia Wen††, Zhihua Zhao†, Hongbo Liu† and Nanchang Xie*

Abstract

**Background:** Autoimmune encephalitis (AE) with multiple auto-antibodies is of great clinical significance because its complex clinical manifestations and atypical imaging increase the difficulty of diagnosis, differential diagnosis and treatment, which may aggravate the disease, increase the recurrence rate and mortality. The coexistence of anti-Leucinie-rich Glioma Inactivated 1 (LGI1) and anti-γ-aminobutyric acid-beta-receptor 1 (GABA<sub>B</sub>R1) has not been published before.

**Case presentation:** We herein present the case of a 60-year-old man with slow response, behavioral changes, psychosis and sleep disorders. Laboratory test included serum hyponatremia, positive serum LGI1 and GABA<sub>B</sub>R1 antibodies using transfected cell-based assays. Electroencephalogram exhibited moderate diffusion abnormality. The patient responded well to steroid impulse treatment and sodium supplement therapy, and did not recur during the follow-up.

**Conclusions:** Here we report the first AE characterized by positive LGI1 and GABA<sub>B</sub>R1 antibodies, as well as summarizing AE with multiple auto-antibodies reported so far, hopefully to provide experience for clinical practice.

**Keywords:** Multiple auto-antibody, Autoimmune encephalitis, Anti-LGI1, Anti-GABA<sub>B</sub>R1

Background

There are basically two kinds of auto-antibodies related to autoimmune encephalitis (AE). One is against neuron surface receptor, among which anti-N-methyl-D-aspartic acid receptor (anti-NMDAR) is the most common, others also including anti-γ-aminobutyric acid-beta-receptor (anti-GABA<sub>B</sub>R), anti-contactin associated protein-like 2 (anti-CASPR2), anti-α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor (anti-AMPAR), anti-Leucinie-rich Glioma Inactivated 1 (anti-LGI1), etc. The other kind is against neuronal intracellular antigen, mainly referring to classic paraneoplastic neuropathy antibody, such as anti-Hu, etc. [1, 2]. The majority of AE patients have only one of the above auto-antibodies, and very few have multiple auto-antibodies.

Different types of auto-antibodies correspond to specific neurological syndrome, which has strong specificity or directivity for etiological diagnosis. Both LGI1 and GABA<sub>B</sub>R1 are autoantigens of treatment-response limbic encephalitis, whose clinical manifestations include the rapid development of mood changes, depression, anxiety and dramatic loss of short-term memory [3]. LGI1-AE is characterized by confusion, cognitive impairment, sleep disorder, refractory hyponatremia, fascio-brachial dystonic seizures and high signal in medial temporal lobe and hippocampus [4]. The symptoms of GABA<sub>B</sub>R1-AE include cognitive dysfunction, seizures and abnormal behavior [5]. The simultaneous occurrence of both antibodies has not been reported before. Herein we report a case of a 60-year-old man with positive anti-LGI1 and anti-GABA<sub>B</sub>R who improved greatly after steroid therapy.
We aim to remind physicians of this rare AE case with multiple auto-antibodies in potential clinical context.

Case presentation
A 60-year-old Chinese male has developed slow response, abnormal behavior and sleep disorder for 1 month. At first, after admitted to the hospital in his hometown and given only sodium supplement and support treatment, his symptoms disappeared but quickly reoccurred. After that, his symptoms became more and more serious and he gradually developed seizures and irritability. He demonstrated confusion, memory loss, insomnia and abnormal behavior when transferred to our hospital in April 2020.

He had no particular previous medical history except for typhia 40 years ago and recovered with no sequel. Neurological exam revealed poor mental state, slow response and damaged memory, attention, calculation and orientation. Cranial nerves, cerebellar function, motor system, sensory system, deep tendon reflexes and pathological reflexes remained normal.

Serum sodium was 119 mmol/L (reference range: 135 ~ 153 mmol/mL) and chlorine was 81 mmol/L (reference range: 90 ~ 110 mmol/L) at first admission. Serum procalcitonin was 0.048 ng/mL (reference range: < 0.046 ng/mL), C reaction protein was 8.05 mg/L (reference range: < 5 mg/L). Cerebrospinal fluid (CSF) electrophoresis IgG index was 0.71 (reference range: 0.3 ~ 0.7). Intracranial pressure was 150 mmH2O. CSF routine biochemistry for protein content and glucose were normal and infectious test for virus, including herpes simplex virus, tuberculosis, fungal and Cryptococcus were negative. CSF cytology and cytometry were negative for malignant cells. Serum AE antibody spectrum demonstrated positive anti-LGI1 IgG and anti-GABAB1R1 IgG using cell-based assays, while other AE-related auto-antibody, such as anti-NMDAR, anti-AMPAR1, anti-AMPAR2, anti-CASPR2 were negative (Fig. 1). Mini mental state examination score was 15. Electroencephalogram (EEG) indicated moderate diffusion abnormality (Fig. 2A and B). Brain enhanced MRI scan was normal. Tests for screening malignancy, including tumor markers and an ultrasound of the liver, gallbladder, spleen, pancreas, kidney, testicle were normal. Chest enhanced CT scan revealed mild inflammation in left lower lobe.

For treatment of AE with coexistent anti-LGI1 and anti-GABAB1R1, he received 1 g and 0.5 g intravenous methylprednisolone separately, 3 days for each dosage, and then remained on an oral steroid taper for half year. After intravenous and oral sodium supplement, blood sodium and chlorine gradually increased to normal (Table 1). His symptoms improved greatly and EEG recovered to normal.

Discussion and conclusion
Here we report the first case of AE with coexistent serum anti-LGI1 and anti- GABAB1R1. The 60-year-old male, with subacute onset, mainly manifested cognitive decline, behavioral abnormality, insomnia, refractory hyponatremia, abnormal EEG, and positive anti-LGI1 and anti-GABAB1R1 in serum. According to research and clinical guidelines [6], the cognitive decline, behavioral abnormality and seizures of the patient are the common clinical manifestations of LGI1-AE and GABAB1R1-AE, but sleep disorder is more common in LGI1-AE, and hyponatremia is unique to LGI1-AE. So, the clinical characteristics of

![Fig. 1](image1)

**Fig. 1** AE-related auto-antibodies in serum measured by cell-based assays. A NMDA; B AMPA1; C AMPA2; D CASPR2; E LGI1; F GABA R1. Anti-LGI1 and Anti-GABA_R1 were positive.
this case were more inclined to anti-LGI1 encephalitis. The patient responded well to glucocorticoid treatment, and we will continue to follow up the prognosis.

LGI1 is a secretory protein mainly expressed in the hippocampus and neocortex, that connects presynaptic epilepsy-related ADAM23 to postsynaptic ADAM22. Anti-LGI1 interrupts the inhibitory signal transmission from the presynaptic potassium channel to the postsynaptic AMPA receptors, thus increasing the excitability of nerve tissue and resulting epilepsy or encephalitis. LGI1-AE, mostly found in elderly men with subacute onset, can demonstrate cognitive impairment, behavioral change, personality abnormality, hyponatremia and frequent seizures, characterized by facio-brachial dystonic seizures, while the majority of patients does not present with generalized tonic-clonic seizures. Most patients have no related tumors, only about 10% had thymoma, while other tumors were rare. Up to 75% of cases have normal CSF routine analysis. EEG can show mild diffuse slow wave, and about half may have swelled medial temporal lobe with high T2/flair signal. The good news is the relatively low recurrence rate [7, 8]. GABA_B_R regulates

Table 1  Blood test for sodium and chlorine

| Date/time | Blood sodium(mmol/L) | Blood chlorine(mmol/L) |
|-----------|----------------------|------------------------|
| 2020-04-21 11:00 | 119.0                | 81.0                   |
| 2020-04-21 20:00 | 121.9                | 82.0                   |
| 2020-04-22    | 124.0                | 85.0                   |
| 2020-04-23    | 129.0                | 92.0                   |
| 2020-04-24    | 135.0                | 93.0                   |
| 2020-04-26    | 138.0                | 92.0                   |
| 2020-04-29    | 134.0                | 91.0                   |
| 2020-04-30    | 135.0                | 91.0                   |

Fig. 2  A  EEG of the patient before treatment (2020-04-21): moderate diffusion abnormality, Wide range of slow waves occur in medium-high waves. B EEG of the patient after treatment (2020-04-29): normal
## Table 2  Clinical data of AE cases with multiple auto-antibodies

| N. | Sex, age | AE auto-Abs | Other Abs | Clinical manifestations | Brain MRI | tumor | prognosis |
|----|----------|-------------|-----------|------------------------|-----------|-------|-----------|
|    |          | serum       | CSF       |                        |           |       |           |
| 1  | M,62     | GABA\textsubscript{A}R | GABA\textsubscript{A}R | Hu | Memory loss, somnolence, concussion, cough, hoarseness | Normal | Lung cancer | Improve |
| 2  | M,61     | GABA\textsubscript{A}R | GABA\textsubscript{A}R | Hu | Epilepsy, somnolence, memory loss | Normal | Lung cancer | Improve |
| 3  | M,59     | GABA\textsubscript{A}R | GABA\textsubscript{A}R | Hu | Epilepsy, psychosis | Lesions of bilateral hippocampus | Lung cancer | Improve |
| 4  | M,58     | GABA\textsubscript{A}R | GABA\textsubscript{A}R | Hu | Psychosis, memory loss, numbness of limbs | Lesions of bilateral hippocampus | Lung cancer | Improve |
| 5  | M,61     | GABA\textsubscript{A}R | GABA\textsubscript{A}R NMDAR | Hu | Epilepsy, memory loss, coma | ND | Lung cancer | Improve |
| 6  | F,19     | –           | NMDAR AQP4 | AQP4 | Psychosis, memory loss, blepharoptosis | Lesions of bilateral basal ganglia, brainstem | No | Improve |
| 7  | F,40     | LGI1 CASPR2 | LGI1 | – | Myalgia, fasciculation, epilepsy, insomnia | Normal | No | Improve |
| 8  | F,56     | LGI1 | LGI1 | Yo | Memory loss, concussion, somnolence, polyphagia | Normal | Thymoma | Improve |
| 9  | F,50     | AMPAR | AMPAR | CV2 | Memory loss, psychosis | Lesions of bilateral cortex | Mediastinal occupying | Dead |
| 10 | F,51     | AMPAR | AMPAR | Hu | Psychosis, dysphagia, dysdiapisia | – | No | Improve |
| 11 | M,44     | ND | GABA\textsubscript{A}R NMDAR | – | Limbic encephalitis | Not mentioned | No | Complete improve |
| 12 | F,63     | – | GABA\textsubscript{A}R | GAD65 | Status epilepticus | Not mentioned | No | Dead |
| 13 | M,60     | GABA\textsubscript{A}R | GABA\textsubscript{A}R | SOX1* | Limbic encephalitis | Not mentioned | SCLC | Partial recovery |
| 14 | M,62     | GABA\textsubscript{A}R | GABA\textsubscript{A}R | Ri* | Limbic encephalitis | Not mentioned | SCLC | – |
| 15 | F,68     | GABA\textsubscript{A}R | GABA\textsubscript{A}R | SOX1* | Limbic encephalitis | Not mentioned | SCLC | Partial recovery |
| 16 | M,74     | GABA\textsubscript{A}R | ND | SOX1 | Limbic encephalitis | Not mentioned | SCLC | Dead |
| 17 | M,77     | GABA\textsubscript{A}R | GABA\textsubscript{A}R | Amphiphysin* | Limbic encephalitis | Not mentioned | SCLC | Unresponsive |
| 18 | F,57     | LGI1 NMDAR | – | – | Faciobrachial dystonic seizure, hyponatremia, mental disorder | Lesions of bilateral cortex | No | Improve |
| 19 | M,66     | GABA\textsubscript{A}R R | GAD* | – | Seizures, confusion | Normal | SCLC | Not available |
| 20 | M,47     | GABA\textsubscript{A}R R | SOX1 VGKC | – | Seizures, behavior change, memory impairment | Bilateral temporal lesions | SCLC | Partial recovery |
| 21 | M,70     | GABA\textsubscript{A}R R | GAD* | SOX1 | Seizures, memory impairment, confusion | Normal | SCLC | Unrespon- sive, dead |
| N. | Sex, age | AE auto-Abs | Other Abs | Clinical manifestations | Brain MRI | tumor | prognosis |
|----|----------|-------------|-----------|------------------------|-----------|-------|-----------|
|    |          | serum       | CSF       |                        | serum     | CSF   |           |
| 22 | M, 58    | GABA<sub>γ</sub>R<sup>+</sup> | Hu<sup>+</sup> | Seizures, memory impairment | Bilateral temporal lesions | SCLC | Unresponsive, dead |
| 23 | M, 61    | GABA<sub>γ</sub>R<sup>+</sup> | BRSK2<sup>+</sup> | Memory impairment | Bilateral temporal lesions | SCLC | Unresponsive |
| 24 | F, 57    | GABA<sub>γ</sub>R<sup>+</sup> | GAD<sup>+</sup> | Subacute cerebellar ataxia | Normal | Carcinoid of thymus | Complete recovery |
| 25 | F, 30    | NMDAR | NMDAR | Epilepsy, psychosis, insomnia | Normal | No | Improve |
| 26 | F, 43    | LGI1 | CASPR2 | Seizures, weight loss, calculation/memory/speech disorder | Bilateral hippocampus/occipital/parietal lesions | No | Improve |
| 27 | F, 67    | LGI1 | Hu | Memory loss, motor aphasia | Left frontal/temporal/occipital/parietal lobe | No | Improve |
| 28 | F, 57    | NMDAR | LGI1 | Seizures, facio-brachial dystonic seizures, somnolence | Demyelination of white matter | Probable lung cancer, thyroid nodule | Improve |
| 29 | F, 84    | GABA<sub>γ</sub>R | Hu | Seizures, cognitive impairment, confusion, psychosis | Left hippocampus and temporal lobe | Probable lung cancer | Dead |
| 30 | M, 55    | NMDAR | Ma2 | Sensory aphasia, memory loss | Left temporal/occipital lobe and hippocampus | No | Improve |
| 31 | F, 60    | GABA<sub>γ</sub>R | amphiphsin | Seizures, cognitive impairment, memory loss | Bilateral hippocampus, right temporal lobe | SCLC | Dead |
| 32 | M, 67    | LGI1 | LGI1 | Seizures, psychosis, memory loss, hand groping, hypotension | Unable to cooperate | No | Improve |
| 33 | F, 43    | NMDAR | Yo | Phychosis, seizures, memory loss | Bilateral hippocampus and temporal lobe | Myoma of uterus | Improve |
| 34 | F, 82    | GABA<sub>γ</sub>R | GABA<sub>γ</sub>R | amphiphsin | Memory loss, psychosis, disorientation | Left temporal lobe and hippocampus | Breast cancer | Unchanged |
voltage-sensitive calcium channel and inward compensatory potassium channels through G protein. GABA$_{\beta}$R is widely spread in brain and spine and particularly abundant in the hippocampus, thalamus and cerebellum. Anti-GABA$_{\beta}$R is related to seizures, memory loss, anxiety and mood disorder. GABA$_{\beta}$R1-AE mostly presents limbic encephalitis symptoms, with temporal lobe epilepsy as the core symptomatology, and most of them are accompanied by cognitive function decline, personality change and mental behavior abnormality. About 50% of patients have small cell lung cancer or neuroendocrine tumor. It is suggested that anti-GABA$_{\beta}$R encephalitis should further take chest CT or PET examination [9].

The overlying of neuronal auto-antibodies may cause the superposition of clinical syndrome, but not a simple complete superposition, which needs to be analyzed according to the specific antibody type and clinical manifestation. According to Professor Guan Hongzhi's newly published review, it is necessary to distinguish whether the antibodies in patients belong to pathogenic markers or concomitant antibodies [10]. The main manifestations of this case are psychobehavioral abnormality and hyponatremia, more similar to clinical manifestations of anti-LGI1 AE.

The co-existence of multiple auto-antibody is rare (summarized in Table 2). Ren Haitao reported 531 cases of AE with auto-antibodies, and only 10 cases detected multiple anti-neuronal antibodies, among whom 5 cases were anti-GABA$_{\beta}$R/anti-Hu, 1 anti-NMDAR/APQ-4, 1 anti-LGI1/anti-CASPR2, 1 anti-LGI1/anti-Yo, 1 anti-AMPAR/anti-CV2 and 1 anti-AMPAR/anti-Hu [10]. In the 20 anti-GABA$_{\beta}$R-AE cases reported by Hoftberger, 7 detected multiple auto-antibodies, among whom 3 cases with anti-Sox1, 1 with anti-Ri, 1 with anti-ampiphysin, 1 with anti-GAD65 and 1 with anti-NMDAR [11]. Liu XY recently reported one case characterized by double positive of anti-LGI1 and anti-NMDAR [12]. Boronat reported a case of anti-GABA$_{\beta}$R combined with anti-GAD65, manifesting cerebellar ataxia and thyroid carcinoid [13]. Qi Hengchang reported two cases of AE with multiple auto-antibodies against neuron (one was anti-NMDAR/anti-GABA$_{\beta}$R, and the other anti-LGI1/anti-CASPR2. Both patients were adult women with acute onset. Their first symptom was epilepsy, and the treatment effect was good [14]. Wang XI retrospectively analyzed 255 AE patients from our hospital and found 7 cases with multiple autoantibodies [15]. Also, Qiu ZD reported 6 AE with coexistent autoantibodies out of 134 cases [16]. Chung recently described a patient with antibodies to GABAB and IgLON5, who presented with sleep disorders like our case [17].

The clinical significance of multiple auto-antibody has already raised attention of many neurologists and needs to be interpreted in combination with clinical practice. For example, anti-GABA$_{\beta}$R can be combined with anti-Hu. When anti-GABA$_{\beta}$R is positive, it is recommended to screen anti-Hu and carry out tumor screening at the same time, such as chest CT, tumor markers, etc., excluding the possibility of tumor as much as possible. In this case chest enhanced CT scan didn’t find tumor, but the patient was advised to take regular examination during follow-up. However, since it’s a single case report, it might be a coincidence despite its great significance.

Here we first report an AE case with co-existing anti-LGI1 and anti-GABA$_{\beta}$R1. The existence of concomitant autoantibodies should be considered when the patients

| Table 2 (continued) |
|---------------------|
| N. | Sex, age | AE auto-Abs | Other Abs | Clinical manifestations | Brain MRI | tumor | prognosis |
|-----|----------|-------------|-----------|------------------------|-----------|-------|----------|
|     |          | serum       | CSF       | serum                  | CSF       |       |          |
| 35  | F,62     | SOX1 Titin  | SOX1      | Polyneuropathy, subacute cerebellar degeneration | –         | Cholangiocarcinoma | Dead     |
| 36  | M,72     | GABA$_{\beta}$R | GABA$_{\beta}$R | Subacute sensory neuropathy, seizures, psychosis | –         | Probable gastric cancer | Dead     |
| 37  | M,62     | LGI1        | LGI1      | Memory loss, seizures, dizziness | Bilateral hippocampus | –       | Improve  |
| 38  | M,70     | LGI1        | CASPR2    | Weekness of limbs, cognitive impairment, baryalia | –         | Probable carcinoid of thymus | Improve  |
| 39  | M,62     | Hu, Ri      | Hu, Ri    | Polyneuropathy | –         | SCLC   | Dead     |
| 40  | M,58     | IgLON5      | GABA$_{\beta}$R | Dysartria, gait  instability, apraxia, hallucination, sleep disorder | Normal   | No     | Improve  |

*Only serum was tested for auto-antibodies, and CSF was not detected*
exhibit atypical and overlapping symptoms. Caution should be given because it’s a single-case report. With the discovery of more multiple auto-antibody positive cases of AE, it will provide evidence for further revealing the clinical characteristics, treatment and prognosis.

Abbreviations

LGI1: Leucine-rich Glioma Inactivated 1; GABABR1: γ-aminobutyric acid-beta-receptor; NMDAR: N-methyl-D-aspartic acid receptor; AMPAR: α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor, CSF: Cerebrospinal fluid; AE: Autoimmune encephalitis.

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Authors’ contributions

XY and WJ together collected disease history and radiological data and drafted this manuscript. XNC designed the manuscript. ZHZ and LHB performed data acquisition. All authors have read and approved the manuscript, and ensure that this is the case.

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Availability of data and materials

There are no associated datasets for this manuscript. All data generated or analyzed during this study are included in this published article. Related queries can be directed to the corresponding author.

Declarations

Ethics approval and consent to participate

Protocols were established, according to the ethical guidelines of the Helsinki Declaration and was approved by the Independent Ethics committee, First Affiliated Hospital of Zhengzhou University. Written informed consent was obtained from the patient.

Consent for publication

Written informed consent regarding the submission and potential publication of this manuscript was obtained from the patient. Additionally, consent for treatment was likewise obtained in the usual fashion during the course of the patient’s hospitalization.

Competing interests

All authors claimed that there were no conflicts of interest. The above funding bodies played a role in the analysis and interpretation of data.

Author details

1 Department of Neurology, The First Affiliated Hospital of Zhengzhou University, Zhengzhou 450052, China. 2 Department of Orthopedics, The First Affiliated Hospital of Zhengzhou University, Zhengzhou 450052, China.

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