

## CASE REPORT

# The psychiatric symptoms in anti-IgLON5 disease: Case report and literature review

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**Key Clinical Message**

Immunotherapy may be ineffective in the advanced stages of anti-IgLON5 disease with psychiatric symptoms. The psychiatric symptoms in advanced stages of anti-IgLON5 disease may be associated with neurodegeneration.

**KEYWORDS**

anti-IgLON5 disease, case report, immunotherapy, neurodegeneration, psychiatric symptoms

## 1 | INTRODUCTION

Recent studies describing patients with autoantibodies against IgLON5, a neuronal cell-adhesion protein, revealed a distinctive sleep disorder, obstructive sleep apnea, gait instability, movement disorders, and brainstem involvement.<sup>1,2</sup> Neuropathological examination demonstrated accumulation of abnormal hyperphosphorylated tau proteins, including 3R and 4R tau isoforms, and neuronal tau deposits in the hypothalamus, the prehypothalamic region, and the tegmentum of the brainstem.<sup>1,3</sup> The HLA-DRB1\*10:01 and HLA-DQB1\*05:01 alleles have been associated with anti-IgLON5 disease, with this association 36-fold higher than in the general population.<sup>4</sup> Moreover, patients with this disease partly responded to immunotherapy.<sup>4-6</sup> These findings indicated that anti-IgLON5 disease involves a complex interaction between neurodegenerative and autoimmune mechanisms, with a genetic predisposition.<sup>4</sup> It remains uncertain whether

anti-IgLON5 disease is an immune disease or a degenerative disease of the nervous system.<sup>3,7-9</sup>

Core symptoms of autoimmune encephalitis include abnormal psychiatric behaviors, cognitive impairment, speech dysfunction, seizures, dyskinesia, autonomic dysfunction, central hypoventilation, and sleep disorders.<sup>10-12</sup> Anti-IgLON5 disease differs from autoimmune encephalitis syndromes because of its protracted disease course, deposition of tau, and variable effect of immunotherapy, making it challenging to diagnose and treat.<sup>3</sup> A previous study found that IgLON5 antibodies were not common in a population of patients with first-episode psychosis or a clinical high-risk for psychosis.<sup>13</sup> However, anti-IgLON5 disease can also have psychiatric symptoms, with these symptoms appearing at onset in some cases, and during the course of disease in others.<sup>14</sup> Few previous studies had summarized and analyzed the psychiatric symptoms of anti-IgLON5 disease. Here, we presented a case with psychiatric symptoms in the advanced stage and reviewed

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relevant literature to explore the clinical manifestations, treatment, and outcomes of anti-IgLON5 disease with psychiatric symptoms.

## 2 | CASE PRESENTATION

A 63-year-old man came to our hospital with the first-presenting psychiatric symptoms, including confusion (Disorder of thinking, behavior, and language), rambles, grabbing things aimlessly, and relieving himself at will (Do not go to the toilet to urinate). At 5 years earlier, the initial symptoms were mild dysphagia, mild dystonia, and sleep disturbances (shouting and sleep attacks, which disappeared when waking up). Although the patient had seen the doctor several times, patient's disease remained undiagnosed. Ten months ago, restriction of neck rotation appeared. Moreover, the bulbar dysarthria, oculomotor dysfunction (restriction of vertical and horizontal gaze), and mild cognitive impairment (Mini Mental State Examination was 21) appeared 4 months ago (Figure 1A).

Neurological examination revealed horizontal and vertical gaze palsy, restricted mouth opening, reduced gag reflex, dysarthria, restriction of neck rotation, mild dystonia of both lower extremities, active tendon reflexes in the extremities, and Babinski's sign in his lower-right extremity. Blood test results showed that immunity, comprehensive metabolism, endocrine, and infection were normal. Blood gas analysis was normal. Brain and brainstem magnetic resonance imaging (MRI) were normal. Tumors were ruled out using chest and abdomen computed tomography and nasopharyngeal-region MRI. His electroencephalogram study was normal but electromyography revealed extensive neurogenic damage. Cerebrospinal fluid (CSF) examination was performed because of the psychiatric symptoms. CSF tests revealed normal nucleated cells and protein. Infectious-disease studies showed negative results for virus, cryptococcus, tuberculosis, bacteria, and fungi. Investigations of paraneoplastic and autoimmune antibodies were performed by collecting antibodies from the patient's serum and CSF, included antibodies to LGI, GABAB receptor, NMDA receptor, Caspr2, AMPA1 receptor, AMPA2 receptor, DPPX, IgLON5, GAD65, GlyR1, DRD2, mGluR5, Hu, Yo, Ri, CV2, Ma2, amphiphysin, Ma1, SOX1, Tr (DNER), Zic4, GAD65, PKC $\gamma$ , recoverin, and titin. Only the anti-IgLON5 antibody test was positive in both CSF and serum (1:320 and 1:100, respectively; Figure 1B).

The patient was started on treatment with intravenous immunoglobulin (IVIg) (2g/kg for 5 days) followed by high-dose steroid therapy (1g/day for 5 days and gradually reduced). Olanzapine was used temporarily when psychiatric symptoms were severe in the initial stages.

One month later, only the abnormal psychiatric behaviors showed any improvement. The patient's other symptoms became worse during the 34 weeks of follow-up.

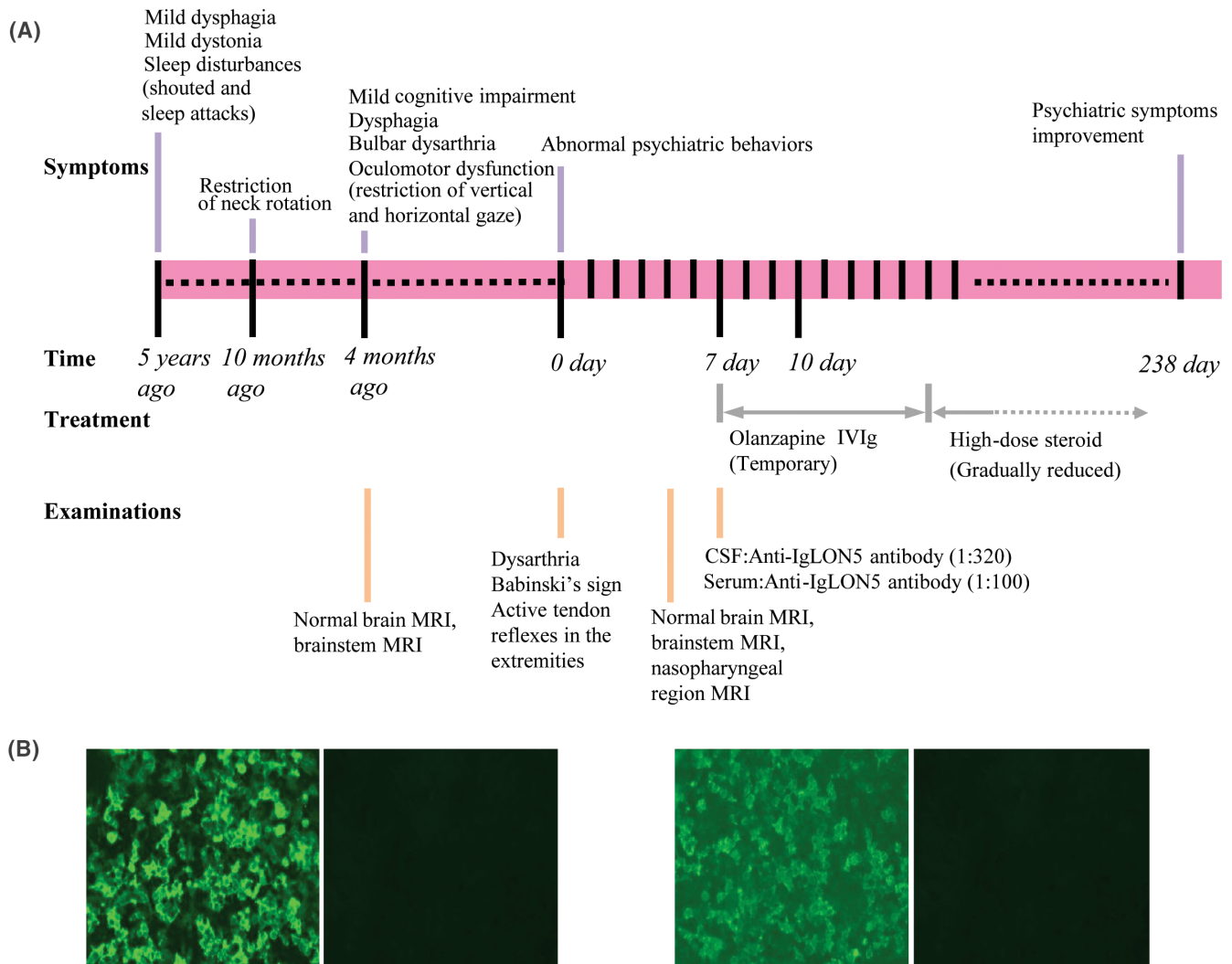
## 3 | REVIEW OF PREVIOUSLY REPORTED CASES OF ANTI-IGLON5 DISEASE WITH PSYCHIATRIC SYMPTOMS

Including our patient, 19 cases were eligible for our review (Table 1; Data S1, Figure S1). Of those, nine patients were male and 10 were female, and the age at diagnosis ranged from 45 to 84 years (average 65 years). The average time from symptom onset to diagnosis was 27 months. For CSF testing, 18 patients had anti-IgLON5 antibody in CSF, and 14 patients had anti-IgLON5 antibody with their serum tested. Of the 16 patients, six had abnormal nucleated cells and/or protein in CSF. Moreover, of 11 patients, three displayed abnormal brain MRI. In terms of psychiatric symptoms, nine patients displayed confusion, seven patients displayed depression, six patients had hallucinations, two patients had delirium, three patients displayed abnormal behaviors, and one patient displayed anxiety. The average time of psychiatric symptoms present was 13 months (range 0–60 months). Among the 14 patients who had received immunotherapy (including steroids, IVIg, plasma exchange, or immunity inhibitors), six partially improved, one fully improved, and seven had no improvement.

## 4 | DISCUSSION

Anti-IgLON5 disease is mainly characterized by sleep disorders, bulbar dysfunction, gait instability, obstructive sleep apnea, movement disorders, oculomotor dysfunction, and neuropsychiatric symptoms.<sup>3,14</sup> Our patient also displayed bulbar dysarthria, dysphagia, oculomotor dysfunction, sleep disturbances, movement disorder, and restriction of neck rotation, with definite abnormal physical signs in the nervous system. Antibody testing was performed because of the psychiatric symptoms, and anti-IgLON5 disease was finally diagnosed. Only the abnormal psychiatric behaviors improved after treatment with IVIg, high-dose steroids, and olanzapine.

Patients with autoimmune encephalitis often had abnormal psychiatric behaviors, including decreased or altered level of consciousness, lethargy, or personality change,<sup>11</sup> and the abnormal psychiatric behaviors often present at an early stage. However, our patient developed psychiatric symptoms after 60 months, and the patient's symptoms were not diagnosed in the past 5 years because the neurological symptoms were mild. Our literature



**FIGURE 1** Clinical characteristics of our patient with anti-IgLON5 disease. Upper part of A shows the symptoms; middle shows the time and the treatment, and lower part shows the examination. Only the abnormal psychiatric behaviors were improved with steroid, IVIg, and olanzapine treatment. The left part of B shows the immunoactivity of the anti-IgLON5 antibodies in CSF (1:320), and the right part shows anti-IgLON5 antibodies in serum (1:100) (original magnification  $\times 200$ ).

review found that the psychiatric symptoms of anti-IgLON5 disease not only present at onset, but during the course of the disease. Therefore, the psychiatric symptoms of anti-IgLON5 disease are different from autoimmune encephalitis syndromes, patients with neurological symptoms and late onset of psychiatric symptoms need to consider anti-IgLON5 disease.

Research had demonstrated that neuronal 3R- and 4R-tauopathy mainly involves the brainstem and hypothalamus in anti-IgLON5 disease.<sup>1,2,15</sup> Both A $\beta$ (1–42) and pathogenic P-tau significantly induced learning and memory deficits and enhanced anxiety behavior in adult male rats.<sup>16</sup> Tau-P301L transgenic mice showed decreased cognitive flexibility in the morris water maze, decreased exploratory behavior, and increased anxiety-like behavior.<sup>17</sup> Participants with elevated tau were twice as likely to be depressed in previous study.<sup>18</sup> Moreover, hallucinations

are also associated with more severe cognitive impairment in Alzheimer's disease,<sup>19</sup> and can be visual or auditory.<sup>20</sup> A previous study revealed that increased baseline hallucinations, apathy, and anxiety were associated with current and future disease progression in Alzheimer's disease.<sup>19</sup> Our literature review also found that the most of patients (13 of 19) with psychiatric symptoms had cognitive impairment (Table 1). Literature review also showed that some patients with psychiatric symptoms had normal nucleated cells and protein in CSF. Our patients also had normal nucleated cells and protein in CSF. Moreover, brain and brainstem magnetic resonance imaging were normal in our study. Our literature review also displayed that the brain MRI of some patients were normal. A previous study found that cultures exposed to anti-IgLON5 antibodies showed reduced neuronal spike rate and synaptic protein content, and a higher proportion of neurons with

TABLE 1 Clinical features of anti-IgLON5 disease with psychiatric symptoms<sup>a</sup>.

No.	Age/sex	Time from symptoms onset to diagnosis (month)	Time of psychiatric symptoms present (month)	Psychiatric symptoms	Clinical presentation	Brain MRI/brain PET-CT	Antibody status in serum	Antibody status in CSF
1	61/M	60	Advanced stages	Paroxysm confusion during daytime, visual hallucinations	Sleep disorder, bulbar dysfunction, obstructive sleep apnea, seizures	Normal brain MRI Brain PET-CT: hypermetabolism of left frontal, temporal lobe, bilateral caudate nucleus and putamen	Positive	Positive
2	59/F	6	9	Confusion	Sleep disorder, bulbar dysfunction, gait disorder, paroxysmal dyspnea, cognitive impairment, movement disorder	Normal brain MRI	Positive	Positive
3	84/F	NA	18	Depression	Obstructive sleep apnea, bulbar dysfunction, oculomotor dysfunction, movement disorder, gait disorder, cognitive impairment	Brain MRI: focal enhancement of leptomeninges	Positive	NA
4	62/M	2	2	Abnormal behaviors (irritability, aggressive behavior, swearing, urination into his slippers)	Sleep disorder, bulbar dysfunction, movement disorder, cognitive impairment	Normal brain MRI	NA	Positive
5	70/F	12	12	Depression	Sleep disorder, gait disorder, movement disorder	Normal brain MRI Brain tau PET-CT: Tau deposition in cerebellar hemispheres, midline cerebellar areas, brainstem areas	Positive	Positive
6	68/F	12	NA	Depression	Sleep disorder, gait disorder, cognitive impairment	Normal brain MRI	Positive	Positive
7	75/F	1	NA	Acute confusion, visual hallucinations	Cognitive impairment, seizures	Brain MRI: frontal subcortical lesions, focal enhancement of leptomeninges	Positive	Positive
8	71/F	72	1	Depression	Sleep disorder, obstructive sleep apnea, gait disorder, movement disorder, cognitive impairment	Normal brain MRI	Positive	Positive
9	45/M	12	0	Confusion	Sleep disorder, obstructive sleep apnea, bulbar dysfunction, gait disorder, cognitive impairment, movement disorder	Normal brain MRI	Positive	Positive

TABLE 1 (Continued)

No.	Age/sex	Time from symptoms onset to diagnosis (month)	Time of psychiatric symptoms present (month)	Psychiatric symptoms	Clinical presentation	Brain MRI/brain PET-CT	Antibody status in serum	Antibody status in CSF
10	69/F	NA	NA	Depression	Oculomotor dysfunction, movement disorder, gait disorder, cognitive impairment	NA	NA	Positive
11	67/F	NA	NA	Delirium, hallucinations	Sleep disorder, bulbar dysfunction, movement disorder, cognitive impairment	NA	NA	Positive
12	70/F	NA	NA	Delirium, depression, hallucinations	Sleep disorder, obstructive sleep apnea, bulbar dysfunction, movement disorder, gait disorder, cognitive impairment, peripheral nervous system	NA	Positive	Positive
13	63/M	NA	NA	Anxiety	Oculomotor dysfunction, bulbar dysfunction, obstructive sleep apnea, peripheral nervous system	NA	NA	Positive
14	61/M	NA	NA	Confusion, hallucinations	Bulbar dysfunction, gait disorder, movement disorder, obstructive sleep apnea, peripheral nervous system	NA	NA	Positive
15	52/M	41	NA	Confusion, visual hallucinations	Sleep disorder, bulbar dysfunction, obstructive sleep apnea, movement disorder, gait disorder	NA	Positive	Positive
16	63/M	18	NA	Recurrent depressive disorder	Sleep disorder, bulbar dysfunction, obstructive sleep apnea, paroxysmal dyspnea, movement disorder, gait disorder	NA	Positive	Positive
17	63/M	60	60	Confusion, gibberish, grabbing things aimlessly, relieve himself at will	Sleep disorder, bulbar dysfunction, oculomotor dysfunction, cognitive impairment, movement disorder	Normal brain MRI	Positive	Positive
18	61/F	1	0	Confusion	Cognitive impairment	Brain MRI: abnormal signals and increased volume of the right hippocampus	Positive	Positive
19	61/M	60	NA	Confusion, body twist, spitting, limbs waving, beating himself	Sleep disorder, bulbar dysfunction, movement disorder	PET-CT: hypermetabolism in left frontal, temporal lobe and bilateral caudate nucleus while hypermetabolism in bilateral putamen.	Positive	Positive

(Continues)

TABLE 1 (Continued)

No.	CSF	HLA-DRBI *10:01; DQBI *05:01 alleles	Neurologic outcome measuring mRS	Therapy	Treatment response	Follow-up (month)
1	Normal	Positive	4	Steroids, IVIg, immunity inhibitor	None	6
2	NA	NA	NA	Steroids, IVIg, plasma exchange	Part	13
3	Increased protein	NA	NA	None	NA	16
4	Pleocytosis and increased protein	Positive	NA	Acyclovir	Part	6
5	Pleocytosis and increased protein	NA	NA	Steroids, immunity inhibitor	Part	12
6	Normal	NA	NA	Steroids, IVIg, immunity inhibitor, plasma exchange	Part	NA
7	Increased protein	Positive	NA	Steroids, immunity inhibitor, plasma exchange	Part	15
8	Pleocytosis and increased protein	Positive	NA	IVIg, immunity inhibitor	None	NA
9	Pleocytosis and increased protein	Positive	NA	Steroids, IVIg, immunity inhibitor	Part	24
10	Normal	NA	6 (death)	None	NA	4
11	Normal	NA	6 (death)	None	NA	6
12	Normal	NA	6 (death)	Steroids	None	19
13	Normal	NA	1	None	NA	9
14	Normal	NA	3	None	NA	129
15	NA	Positive	2	Steroids, IVIg, immunity inhibitor, plasma exchange	None	NA
16	NA	Positive	3	Steroids, IVIg, immunity inhibitor, plasma exchange	None	NA
17	Normal	NA	4	Steroids, IVIg, olanzapine	Part (psychiatric symptoms)	8
18	Normal	Positive	1	IVIg, immunity inhibitor	Full	13
19	Normal	Positive	4	Steroids, IVIg, immunity inhibitor	None	48

Abbreviations: F, female; IVIg, intravenous immunoglobulins; M, male.

<sup>a</sup>The literature we reviewed is presented in Data S2.



degeneration, including p-tau positive neurons, indicating that pathological anti-IgLON5 antibodies induced neurodegenerative changes and cell death in human neurons.<sup>21</sup> Therefore, we speculated that the normal brain MRI and psychiatric symptoms in our patient might be associated with neurodegeneration of anti-IgLON5 disease.

Although some patients with IgLON5 antibodies were responsive to immunotherapy, consistent with most other forms of neuronal cell-surface autoantibody-mediated diseases,<sup>22,23</sup> patients with improvement in symptoms still require long-term follow-up because symptoms may worsen and patients may die suddenly.<sup>1,14,24,25</sup> Grüter et al.<sup>26</sup> found that first-line immunotherapy of relapse-like acute-to-subacute exacerbation episodes resulted in improvement in 41% of patients and early immunotherapy (before advanced neurodegeneration) was associated with a better long-term clinical outcome. In cultures of hippocampal neurons, antibodies of patients caused a decrease in cell-surface IgLON5 clusters that was not reversed after IgLON5 antibodies were removed from the media, indicating an irreversible antibody-mediated internalization of surface IgLON5 in hippocampal neurons.<sup>27</sup> In our patient, only the abnormal psychiatric behaviors showed improvement with immunotherapy, but the patient's other symptoms did not improve. These may suggest neurodegeneration of anti-IgLON5 disease respond poorly to immunotherapy, and it is important to diagnose anti-IgLON5 disease early for patients. The improvement of our patient's psychiatric symptoms may be associated with antipsychotics.

There were several limitations in our study. First, although causes of psychiatric symptoms (e.g., tumor, metabolism, immunity, infection, and endocrine) were excluded in our case, it cannot be fully ruled out that psychiatric symptoms were related to the consequence of other aggravated symptoms. Second, videopolysomnography and detailed neuropsychological assessment were not performed in our study.

In general, the psychiatric symptoms of anti-IgLON5 disease can present not only in the early stages of the disease, but also in the advanced stages. The psychiatric symptoms may be associated with neurodegeneration in our patient. Immunotherapy may be ineffective in the advanced stages of anti-IgLON5 disease. Therefore, it is necessary to consider anti-IgLON5 disease when for patients with psychiatric symptoms and neurological symptoms.

#### AUTHOR CONTRIBUTIONS

**Yuanyuan Luo:** Data curation; formal analysis; investigation; methodology; project administration; writing – original draft; writing – review and editing. **Jun Xiao:** Data curation; project administration. **Jieying Li:** Data curation; project administration.

#### FUNDING INFORMATION

No targeted funding.

#### CONFLICT OF INTEREST STATEMENT

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

#### DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

#### ETHICS STATEMENT

This report was approved by the Ethics Committee of Sichuan Provincial People's Hospital.

#### CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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## SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

**How to cite this article:** Luo Y, Xiao J, Li J. The psychiatric symptoms in anti-IgLON5 disease: Case report and literature review. *Clin Case Rep*. 2024;12:e8310. doi:[10.1002/ccr3.8310](https://doi.org/10.1002/ccr3.8310)