Short Communication
Volume 15:01, 2025

Journal of Clinical Case Reports

ISSN: 2165-7920 Open Access

Autoimmune Limbic Encephalitis Presenting as Late-onset Anxiety Disorder

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Introduction

Autoimmune Limbic Encephalitis (ALE) is a rare but increasingly recognized neuroinflammatory disorder characterized by subacute onset of neuropsychiatric symptoms, including cognitive decline, seizures and mood disturbances. While often associated with paraneoplastic syndromes, ALE can also occur idiopathically and may present without classic neurological features. Anxiety, depression and behavioral changes may be the first signs, especially in older adults. These symptoms can lead to misdiagnosis as a primary psychiatric disorder, delaying appropriate immunotherapy. Late-onset anxiety disorder is uncommon and should prompt evaluation for secondary causes when accompanied by subtle cognitive or neurological signs. This case underscores the importance of a multidisciplinary approach in diagnosing neuroimmune disorders that masquerade as primary psychiatric illnesses. Additionally, this case underscores the importance of clinical vigilance when evaluating psychiatric symptoms in older adults. Unlike primary psychiatric illnesses, which typically emerge earlier in life, autoimmune or neurodegenerative processes often manifest later and follow an atypical or rapidly progressive course [1].

Description

A 61-year-old woman with no psychiatric history presented with a six-week history of progressive anxiety, insomnia and panic attacks. She reported episodic fear, restlessness and occasional visual hallucinations. There was no prior history of depression, substance use, or cognitive complaints. Initial psychiatric evaluation diagnosed her with generalized anxiety disorder and she was started on escitalopram and clonazepam with minimal improvement. Over the next two weeks, she developed short-term memory loss and disorientation. Neurological consultation revealed mild confusion and episodic anomia. MRI brain showed increased FLAIR signal in the bilateral medial temporal lobes. CSF analysis was unremarkable but serum testing revealed positive anti-LGI1 antibodies. A diagnosis of autoimmune limbic encephalitis was made. She was started on high-dose intravenous methylprednisolone followed by oral prednisone taper. Her symptoms improved significantly within three weeks. Repeat MRI showed resolution of temporal hyperintensities. At six-month follow-up, she remained asymptomatic and off psychiatric medication [2].

Autoimmune limbic encephalitis can present insidiously, often mimicking primary psychiatric or cognitive disorders. In elderly patients, new-onset psychiatric symptoms such as anxiety, paranoia, or hallucinations should prompt consideration of an organic etiology. The limbic system's involvement explains the emotional and cognitive symptoms, particularly memory loss and behavioral disturbances. Anti-LGI1 encephalitis, a common subtype of ALE, frequently presents without seizures and may initially lack overt neurological signs, increasing the risk of misdiagnosis. MRI findings of medial temporal lobe hyperintensity are often diagnostic, supported by detection of neuronal surface antibodies in serum or CSF. Immunotherapy with corticosteroids, IVIG, or plasma exchange forms the cornerstone of treatment and can lead to complete resolution if initiated early. Delay in diagnosis may result in irreversible hippocampal atrophy and long-term cognitive deficits [3].

This case highlights the risk of attributing late-onset psychiatric symptoms to functional disorders without adequate investigation. The patient's anxiety and hallucinations were initially misdiagnosed, leading to a delay in appropriate care. Atypical features such as late age of onset, rapid progression and poor response to psychiatric medications are red flags warranting neurological workup. Neuroimaging and antibody testing are essential tools in diagnosing autoimmune encephalitis. Early immunosuppression can not only reverse symptoms but also prevent further neurological damage. Coordination between psychiatry, neurology and immunology is key in identifying and managing these complex cases effectively [4].

Visual hallucinations, in particular, are uncommon in primary anxiety disorders and should raise suspicion of an organic cause, especially in the presence of cognitive decline or fluctuating mental status. While functional psychiatric disorders are more prevalent, ruling out reversible medical causes is crucial before initiating long-term psychotropic therapy in older patients. Furthermore, the presence of anti-LGI1 antibodies in serum rather than CSF—as seen in this patient—is consistent with current understanding of autoimmune encephalitis, where peripheral antibody testing may be more sensitive in some subtypes. This reinforces the need for both serum and CSF antibody panels in suspected cases. Clinicians should also be aware of associated findings such as hyponatremia, which often accompanies LGI1 encephalitis due to syndrome of inappropriate antidiuretic hormone secretion (SIADH). Timely recognition and treatment can lead to dramatic clinical recovery, as demonstrated in this case, while also preventing long-term cognitive morbidity. Continued education and awareness among psychiatrists and primary care providers are essential to improving outcomes in these diagnostically challenging cases [5].

Conclusion

Autoimmune limbic encephalitis is a reversible yet potentially disabling condition that may initially mimic psychiatric illness, particularly in older adults. Clinicians must maintain a high index of suspicion for ALE in cases of late-onset anxiety or behavioral changes with rapid progression and cognitive involvement. Neuroimaging and antibody screening are essential for diagnosis. Prompt immunotherapy leads to favorable outcomes and prevents chronic neurological impairment. This case exemplifies the importance of thorough evaluation in psychiatric presentations that deviate from typical age patterns or resist conventional treatment.

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Received: 02 January, 2025, Manuscript No. jccr-25-167980; **Editor assigned:** 04 January, 2025, PreQC No. P-167980; **Reviewed:** 16 January, 2025, QC No. Q-167980; **Revised:** 23 January, 2025, Manuscript No. R-167980; **Published:** 30 January, 2025, DOI: 10.37421/2165-7920.2025.15.1639

Acknowledgment

None.

Conflict of Interest

None.

References

1. Graus, Francesc, Desamparados Escudero, Laura Oleaga and Jordi Bruna, et al. "Syndrome and outcome of antibody-negative limbic encephalitis." *Eur J Neurol* 25 (2018): 1011-1016.

2. Cohen, Jesse, Javier Sotoca, Shikha Gandhi and Anusha K. Yeshokumar, et al. "Autoimmune encephalitis: A costly condition." *Neurology* 92 (2019): e964-e972.

3. McGinley, Marisa, Sarkis Morales-Vidal and Sean Ruland. "Leucine-rich glioma inactivated-1 and voltage-gated potassium channel autoimmune encephalitis associated with ischemic stroke: A case report." *Front Neurol* 7 (2016): 68.

4. Varley, James A., Magnus Andersson, Eleanor Grant and Antonio Berretta, et al. "Absence of neuronal autoantibodies in neuropsychiatric systemic lupus erythematosus." *Ann Neurol* 88 (2020): 1244-1250.

5. *Rojas, Manuel, Paula Restrepo-Jimenez, Diana M. Monsalve and Yovana Pacheco, et al.* "Molecular mimicry and autoimmunity." *J Autoimmun* 95 (2018): 100-123.

How to cite this article: Ahmad, Moustafa. "Autoimmune Limbic Encephalitis Presenting as Late-onset Anxiety Disorder." *J Clin Case Rep* 15 (2025): 1639.