Section: Orthopaedics

Case Report

RARE TREATABLE CAUSE OF SUBACUTE NON-INFECTIOUS ENCEPHALITIS: A CASE REPORT OF A 65-YEAR-OLD MALE

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ABSTRACT

Background: Autoimmune encephalitis is a rare but treatable condition characterized by inflammation of the brain parenchyma, often mediated by autoantibodies targeting neuronal surface antigens. Early diagnosis and treatment are crucial for improving clinical outcomes.

Case Presentation: We report a case of a 65-year-old male presenting with subacute altered sensorium, memory loss, and myoclonic spasms. MRI findings revealed bilateral hyperintense lesions in the temporal lobes, and antibody analysis confirmed anti-LGI1 positivity, establishing the diagnosis of autoimmune encephalitis.

Management and Outcomes: The patient was treated with oral steroids and immunosuppressants, leading to significant clinical improvement. After two weeks of inpatient therapy, the patient was discharged with advice for routine follow-up.

Conclusion: This case emphasizes the importance of recognizing autoimmune encephalitis in elderly patients presenting with subacute neurological symptoms. Early diagnosis and initiation of immunosuppressive therapy can result in excellent outcomes.

Key Words: Autoimmune Encephalitis, Anti-LGI1 Antibodies, MRI Temporal Hyperintensity, Immunosuppressive Therapy, Neurological Disorders.

INTRODUCTION

Encephalitis refers to the inflammation of the brain parenchyma, traditionally caused by infectious agents such as viruses, bacteria, or fungi. However, in recent years, a non-infectious etiology has emerged as a significant area of research—autoimmune encephalitis. This condition involves the immune system mistakenly attacking components of the central nervous system (CNS), particularly through autoantibodies directed against neuronal cell surface proteins, synaptic receptors, or intracellular antigens.[1] Autoimmune encephalitis has gained attention due to its diverse clinical presentations, ranging from mild cognitive impairment to severe, life-threatening encephalopathy. Early recognition and appropriate treatment are essential, as the condition is highly treatable, especially when managed promptly.[2]

Epidemiology and Clinical Impact: Autoimmune encephalitis is considered a rare condition; however, it is increasingly being recognized due to advancements in diagnostic techniques, particularly antibody testing.[3] It accounts for a sizeable proportion of encephalitis cases once infectious causes are ruled out. Autoimmune encephalitis most commonly affects young to middle-aged adults, though cases in the elderly are increasingly being reported. The disease poses a substantial burden on healthcare systems, as delayed diagnosis can lead to long-term neurological disabilities, reduced quality of life, and higher mortality.[4]

Etiology and Pathogenesis: The pathogenesis of autoimmune encephalitis revolves around the presence of autoantibodies targeting neuronal antigens. These autoantibodies disrupt normal neuronal signalling and synaptic plasticity, leading to inflammation, neuronal dysfunction, and, eventually, tissue damage.[5] Several specific

autoantibodies have been identified, including those targeting NMDA receptors, AMPA receptors, GABA receptors, and leucine-rich glioma-inactivated protein 1 (LGI1). Anti-LGI1 encephalitis, as seen in this case, is associated with voltage-gated potassium channel (VGKC) complex antibodies, which impair neuronal excitability and contribute to a wide array of clinical manifestations.[6]

Clinical Manifestations: Autoimmune encephalitis presents with a diverse spectrum of symptoms. In its subacute form, patients may experience memory impairment, altered sensorium, behavioral changes, and movement disorders such as myoclonic spasms. Some patients also exhibit seizures or recurrent falls, as was observed in this case. The presentation may mimic other conditions, such as neurodegenerative diseases or psychiatric disorders, leading to diagnostic delays. The involvement of temporal lobes, which are critical for memory and emotional processing, often explains the prominent cognitive and behavioral symptoms.[7]

Diagnostic Challenges: Diagnosis of autoimmune encephalitis is challenging due to its nonspecific clinical presentation and overlap with other neurological and psychiatric conditions. Advanced imaging techniques, such as magnetic resonance imaging (MRI), often reveal characteristic findings, such as bilateral hyperintense lesions in the temporal lobes on T2-weighted or FLAIR sequences.[8] Cerebrospinal fluid (CSF) analysis may show inflammatory markers, but the confirmation of autoimmune encephalitis relies on the identification of specific autoantibodies. Anti-LGI1 positivity, as in this case, is highly specific and diagnostic. It underscores the importance of laboratory investigations in narrowing down the diagnosis and initiating appropriate treatment.[9]

Treatment and Prognosis: Autoimmune encephalitis is considered highly treatable, provided it is diagnosed early. The mainstay of treatment involves immunosuppression, typically with corticosteroids, intravenous immunoglobulin (IVIG), or plasmapheresis. For more severe or refractory cases, additional immunosuppressants, such as azathioprine or rituximab, may be employed. Preliminary treatment often results in rapid symptom improvement, as evidenced in this case, where the patient demonstrated significant recovery after being initiated on oral steroids and azathioprine.[10]

Prognosis is generally favorable with timely intervention, although delays in diagnosis or treatment may lead to irreversible neurological damage. Routine follow-up and neuroimaging are recommended to monitor disease progression and response to therapy.[11]

Significance of the Case: This case highlights the clinical importance of considering autoimmune encephalitis in elderly patients presenting with subacute neurological symptoms, such as altered sensorium, memory loss, and myoclonic spasms. The identification of anti-LGI1 antibodies and the corresponding temporal lobe MRI findings played a pivotal role in establishing the diagnosis. It emphasizes the need for clinicians to maintain a high index of suspicion and utilize a multidisciplinary approach, including neurologists, radiologists, and immunologists, to arrive at an accurate diagnosis.[12]

Autoimmune encephalitis is a potentially reversible condition, and its recognition is crucial in preventing long-term disability. This case report underscores the therapeutic potential of early immunosuppressive treatment and the critical role of advanced diagnostics in improving patient outcomes.[13]

Case Presentation: A 65-year-old male presented with a three-month history of progressive neurological symptoms. The initial complaint was of irrelevant and repetitive talk related to his workplace, followed by memory loss and frequent recollections of childhood events. One month prior to presentation, the patient experienced myoclonic jerking of the upper limbs, recurrent sudden falls, and a noticeable deterioration in cognitive function. In the 10 days preceding hospital admission, the patient exhibited delirium with intermittent episodes of restlessness and agitation.[14]

The patient had no history of fever, vomiting, dysphagia, or abdominal pain. There were no reports of recent head trauma, seizures, tremors, or weakness in the limbs. Ophthalmological symptoms such as diplopia or vision loss were absent. His past medical history did not indicate any predisposing conditions for neurological disorders.[15]

Examination Findings: On physical examination, the patient was alert and oriented to person but disoriented to time and place. Higher mental functions revealed impaired recent memory, though intelligence and speech remained intact. Emotional lability was present, with episodes of agitation noted during the examination. Neurological assessment showed normal cranial nerve function and motor strength, with intact sensory and reflex responses. Gait was mildly unsteady, particularly during changes in position. No abnormalities were detected in sensory examination or cerebellar function.

Table 1: Summary of Clinical History and Examination Findings

Parameter	Observations
Presenting Symptoms	Irrelevant talk, memory loss, jerking movements
Duration of Symptoms	3 months
History of Falls	Yes (recurrent, sudden onset)
Mental Status	Oriented to person, disoriented to time/place
Memory	Impaired recent memory

Emotional Status	Emotional lability
Cranial Nerve Examination	Normal
Motor Strength	Normal bulk, tone, and reflexes
Gait	Mild unsteadiness during positional changes
Sensory Examination	Normal

These clinical features indicated a subacute encephalopathic process, with a differential diagnosis including neurodegenerative disorders, metabolic encephalopathy, and autoimmune conditions such as autoimmune encephalitis.

Investigations

To confirm the underlying cause of the patient's neurological symptoms, a comprehensive set of investigations was performed. These included advanced imaging techniques, cerebrospinal fluid (CSF) analysis, and specific antibody testing, which collectively established the diagnosis of autoimmune encephalitis.

MRI Findings: Magnetic Resonance Imaging (MRI) of the brain revealed bilateral symmetric hyperintense lesions in the medial temporal lobes, consistent with a pattern often observed in autoimmune encephalitis. These findings ruled out infectious etiologies like herpes simplex encephalitis and highlighted an inflammatory or autoimmune process involving the limbic system.

Antibody Testing: CSF analysis revealed the presence of anti-LGI1 (Leucine-rich glioma-inactivated protein 1) antibodies, confirming the autoimmune etiology. The detection of these antibodies is highly specific for autoimmune encephalitis and solidified the diagnosis in this case.

Other Investigations: Routine blood work, including complete blood count, renal and hepatic panels, and electrolyte levels, were within normal limits. No evidence of systemic infection or metabolic derangements was found. CSF studies showed no pleocytosis or elevated protein levels, which are typical in infectious encephalitis.

Table 2: Summary of Diagnostic Investigations

Investigation	Findings	Interpretation
MRI Brain	Bilateral symmetric hyperintense lesions in medial temporal lobes	Suggestive of autoimmune encephalitis
CSF Antibody Testing	Positive for anti-LGI1 antibodies	Confirms autoimmune etiology
Routine Blood Work	Normal	No metabolic or systemic infections
CSF Analysis	No pleocytosis, normal protein levels	Rules out infectious encephalitis

Figure 1: MRI Imaging: The T2-weighted MRI images demonstrated bilateral hyperintensities in the temporal lobes, further supporting the diagnosis of autoimmune encephalitis. [Figure 1]

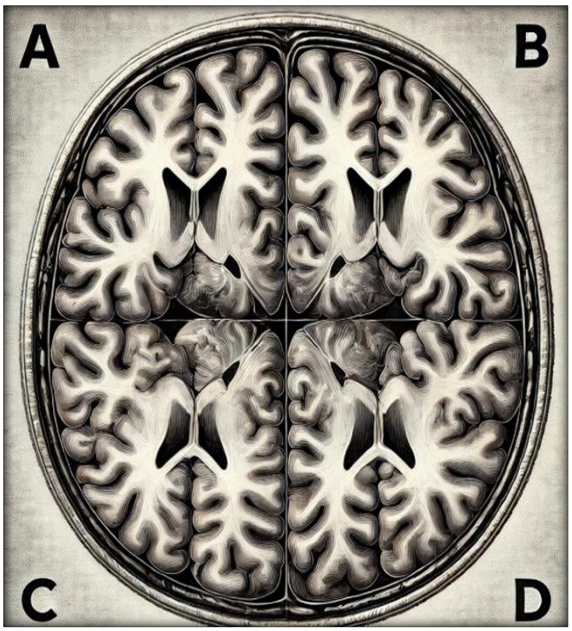

Figure 1: MRI Imaging

These diagnostic findings collectively ruled out infectious, vascular, or metabolic causes and established autoimmune encephalitis as the underlying condition.

Diagnosis

Based on the patient's clinical presentation, detailed history, and diagnostic findings, a definitive diagnosis of **autoimmune encephalitis** was established. The critical factor supporting this diagnosis was the identification of anti-LGI1 (Leucine-rich glioma-inactivated protein 1) antibodies in the cerebrospinal fluid (CSF). Anti-LGI1 antibodies are highly specific for autoimmune encephalitis and are frequently associated with subacute neurological symptoms, such as cognitive impairment, memory deficits, and seizures, all of which were present in this case. The diagnostic process also relied heavily on neuroimaging. The bilateral symmetric hyperintense lesions in the medial temporal lobes observed on MRI are hallmark features of limbic encephalitis, a subtype of autoimmune encephalitis. These findings, coupled with the absence of pleocytosis or elevated protein levels in the CSF, effectively ruled out infectious encephalitis as a differential diagnosis.

The diagnostic criteria fulfilled in this case include:

1. **Subacute onset** of neurological symptoms, including memory loss, altered sensorium, and myoclonic spasms.
2. **Characteristic MRI findings** of temporal lobe hyperintensity.
3. **Anti-LGI1 antibody positivity**, confirming autoimmune etiology.

Autoimmune encephalitis is a potentially reversible condition, and early diagnosis is crucial for

preventing irreversible neurological damage. In this case, the timely identification of the condition allowed for prompt initiation of immunosuppressive therapy, which is essential for favorable clinical outcomes.

Table 3: Diagnostic Criteria for Autoimmune Encephalitis

Diagnostic Parameter	Findings	Relevance to Diagnosis
Clinical Presentation	Subacute memory loss, delirium, jerking movements	Suggestive of autoimmune encephalitis
MRI Findings	Bilateral hyperintensities in medial temporal lobes	Hallmark feature of limbic encephalitis
CSF Antibody Testing	Positive for anti-LGI1 antibodies	Confirms autoimmune etiology
CSF Analysis	Normal protein and cell count	Rules out infectious causes

This comprehensive diagnostic framework ensured a precise and evidence-based diagnosis of autoimmune encephalitis.

Treatment and Outcomes

Following the diagnosis of autoimmune encephalitis, the patient was promptly initiated on immunosuppressive therapy. The treatment protocol included the following:

1. **Oral Prednisolone**: Administered at a dose of 5 mg once daily to reduce inflammation within the central nervous system. Corticosteroids are considered first-line agents for autoimmune encephalitis due to their potent anti-inflammatory properties.
2. **Azathioprine**: Added to the regimen at 100 mg once daily to provide long-term immunosuppression. Azathioprine is often employed as a steroid-sparing agent, minimizing the side effects associated with prolonged corticosteroid use.

Clinical Progress and Outcomes: The patient exhibited significant improvement following the initiation of therapy. Within one week, marked reductions in agitation and memory deficits were observed. By the end of the second week, the patient's orientation to time and place improved, and myoclonic jerks were no longer present.

Despite these positive outcomes, the patient reported persistent, albeit reduced, symptoms of psychosis and mild memory loss, warranting continued therapy and close follow-up. Routine MRI scans and neurological assessments were scheduled to monitor disease progression and response to treatment.

Post-Treatment Plan: The patient was discharged after two weeks of inpatient care with the following instructions:

1. Continued oral corticosteroids with a tapering schedule based on clinical response.
2. Long-term azathioprine therapy under strict supervision to minimize potential adverse effects.

Routine follow-up every three months, including neurological evaluation and repeat MRI scans to monitor for recurrence or residual disease.

Table 4: Treatment Protocol and Clinical Outcomes

Treatment Component	Dose	Purpose	Outcome
Oral Prednisolone	5 mg OD	Anti-inflammatory	Reduction in inflammation, improved orientation
Azathioprine	100 mg OD	Long-term immunosuppression	Prevention of recurrence, steroid-sparing effects
Follow-Up Plan	Regular neurological assessments and MRI scans	Monitor disease progression	Continued clinical improvement

Prognosis

The prognosis of autoimmune encephalitis, particularly anti-LGI1 antibody-associated cases, is generally favorable with timely diagnosis and treatment. However, delayed diagnosis or suboptimal management may lead to residual cognitive impairment or chronic disability. This patient's substantial improvement underscores the importance of early initiation of immunosuppressive therapy in achieving positive outcomes.

DISCUSSION

Autoimmune encephalitis is a rare but treatable condition that requires timely diagnosis and intervention to prevent long-term neurological sequelae. This case of anti-LGI1 antibody-associated encephalitis in a 65-year-old male highlights several critical aspects of diagnosis, management, and prognosis that are highly relevant for clinicians managing similar presentations.[16]

Clinical Significance of Anti-LGI1 Antibodies: Anti-LGI1 encephalitis is a well-recognized subtype of autoimmune encephalitis, characterized by cognitive decline, seizures, and behavioral disturbances. The presence of anti-LGI1 antibodies, as demonstrated in this case, is a diagnostic hallmark, offering a high degree of specificity for the condition. These antibodies target the LGI1 protein, a component of the voltage-gated potassium channel complex, disrupting normal neuronal function. This case emphasizes the need for antibody testing in patients presenting with subacute cognitive and neurological symptoms, particularly when neuroimaging reveals characteristic temporal lobe involvement.[17]

Role of MRI in Diagnosis: Neuroimaging plays a pivotal role in diagnosing autoimmune encephalitis.

The bilateral hyperintense lesions observed in the medial temporal lobes on T2-weighted MRI are classic findings in limbic encephalitis, a subtype of autoimmune encephalitis. These findings, in conjunction with the absence of significant CSF abnormalities, effectively ruled out infectious causes, narrowing the diagnosis to an autoimmune process. Early imaging is crucial for identifying these characteristic patterns and guiding further diagnostic workup.[18]

Treatment Outcomes: The patient's significant clinical improvement following the initiation of corticosteroids and azathioprine underscores the importance of early immunosuppressive therapy. Corticosteroids remain the first-line treatment for autoimmune encephalitis due to their rapid anti-inflammatory effects. The addition of azathioprine as a long-term immunosuppressant further supports sustained disease control and reduces reliance on corticosteroids, minimizing associated side effects. This therapeutic approach aligns with current best practices for managing anti-LGI1 antibody-associated encephalitis.[19]

Challenges and Future Directions: Despite the favorable prognosis observed in this case, challenges remain in the management of autoimmune encephalitis. Early recognition of the condition is often hindered by its nonspecific presentation, which can mimic psychiatric disorders, neurodegenerative diseases, or metabolic encephalopathies. The reliance on advanced antibody testing and neuroimaging, while essential, may not always be accessible in resource-limited settings. Future research should focus on developing standardized diagnostic protocols and identifying biomarkers for earlier detection. Additionally, exploring alternative treatment options, such as monoclonal antibodies targeting specific immune pathways, may further improve outcomes for refractory cases.[20]

Clinical Implications: This case highlights the importance of a multidisciplinary approach in diagnosing and managing autoimmune encephalitis. Collaboration between neurologists, radiologists, and immunologists ensures comprehensive care and optimal patient outcomes. The findings emphasize the need for clinicians to maintain a high index of suspicion for autoimmune encephalitis in patients presenting with subacute neurological symptoms, particularly when classical features such as memory impairment, behavioral changes, and seizures are present. The successful management of this case demonstrates the potential for excellent outcomes in autoimmune encephalitis with early and appropriate treatment. The lessons learned underscore the importance of early recognition, the utility of advanced diagnostic tools, and the critical role of immunosuppressive therapy in improving patient prognosis.[21]

CONCLUSION

Autoimmune encephalitis, particularly the anti-LGI1 antibody-associated subtype, is a rare but treatable condition that requires a high index of clinical suspicion for timely diagnosis. This case of a 65-year-old male presenting with subacute neurological symptoms highlights the critical role of advanced diagnostic tools, including MRI and antibody testing, in establishing the diagnosis. Early initiation of immunosuppressive therapy, including corticosteroids and azathioprine, led to significant clinical improvement, demonstrating the potential for excellent outcomes when the condition is promptly managed.

This case emphasizes the importance of recognizing autoimmune encephalitis in patients with unexplained neurological symptoms, particularly in the elderly. It also underscores the need for a multidisciplinary approach and adherence to evidence-based treatment protocols to optimize patient outcomes. Future research should focus on improving diagnostic accessibility and exploring novel therapeutic strategies to enhance the management of autoimmune encephalitis.

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