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Case Report

Dual association of autoimmune encephalitis with anti-NMDAR and anti-GAD65 antibodies: A Case Report with Literature Review

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ABSTRACT

Anti-N-methyl-D-aspartate receptor (anti-NMDAR) encephalitis is the most common type of autoimmune encephalitis, whereas anti-glutamic acid decarboxylase 65 (antiGAD65) encephalitis is a rare autoimmune condition. The coexistence of these two conditions has been rarely reported. In this article, we will discuss this rare association through a case report and attempt to determine its main characteristics. We report the case of an 18-year-old male with no medical history, admitted to the medical Intensive Care Unit (ICU) with a decreased level of consciousness, bizarre behavior, and abnormal movements for one week. These symptoms followed the progression of initial signs such as delirium, which had begun two months earlier. Laboratory analysis revealed an inflammatory syndrome with rhabdomyolysis. Cerebral angio-MRI findings were unremarkable. The electroencephalogram (EEG) showed slow, non-reactive activity. Cerebrospinal fluid (CSF) analysis and infectious studies were normal. However, immunological testing using the immunofluorescence technique revealed the presence of anti-NMDAR antibodies in both serum and CSF, as well as anti-GAD65 antibodies in the serum. The positron emission tomography (PET) scan screening for neoplasm was negative. Therapeutically, the patient was treated with anticonvulsants, antipsychotics, intravenous immunoglobulins, corticosteroids, plasma exchanges, cyclophosphamide, and rituximab. Consequently, he demonstrated a remarkable gradual clinical improvement. This case highlights an aspect of autoimmune dysregulation that may lead to atypical and severe clinical presentations. The co-occurrence of anti-NMDAR and anti-GAD65 encephalitis is a rare condition that can lead to severe manifestations. Early diagnosis using a broad antibody panel facilitates timely and appropriate management.

1. Introduction

Over the past years, research on autoimmune encephalitis has identified numerous antibodies responsible for different disease subtypes [1]. In a multicenter population-based prospective study conducted in the United Kingdom, anti-N-methyl-D-aspartate receptor (anti-NMDAR) encephalitis accounted for 4% of all causes of encephalitis [2]. In a nationwide cohort study in the Netherlands, anti-NMDAR encephalitis represented 17.5% and anti-glutamic acid decarboxylase 65 (antiGAD65) encephalitis 13.5% of autoimmune encephalitis and paraneoplastic neurological syndromes [3].

Autoimmune encephalitis can present with clinical manifestations of varying severity. A study by Gaspard N et al. reported that autoimmune encephalitis was responsible for 37% of cases of refractory status epilepticus; among these, anti-GAD65 accounted for 2%, and anti-NMDAR encephalitis for 12% [4]. The ICU-CompoSE study (ICU–Complications of Severe Encephalitis) found that anti-NMDAR encephalitis accounted for 62% and anti-GAD65 encephalitis for 6% of patients with autoimmune encephalitis admitted to the ICU. In this study, mechanical ventilation, sepsis, tumor presence, and autonomic dysfunction were associated with prolonged ICU stays or incomplete recovery [5].

Although the co-occurrence of different types of autoimmune encephalitis has been reported, the coexistence of anti-NMDAR and anti-GAD65 encephalitis is rare, with limited data available regarding the severity of this association. Herein, we report the case of an 18-year-old male diagnosed with both anti-NMDAR and anti-GAD65 encephalitis who was admitted to the ICU due to a decreased level of consciousness. The patient received first- and second-line immunotherapies, resulting in a gradual and sustained clinical recovery.

2. Case Presentation

We report the case of an 18-year-old Moroccan male patient with no medical history, admitted to the medical intensive care unit for decreased level of consciousness, abnormal movements, and fever. These symptoms had developed one week prior to admission, following the progression of initial symptoms, including delirium, bizarre behavior, and hallucinations, which had begun two months

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earlier after a flu-like episode. On admission, the patient was febrile with a temperature of 39.2°C and had a Glasgow Coma Scale score of 11. He exhibited mutism, was unresponsive to stimuli, and showed generalized rigidity. Autonomic dysfunctions were also noted, including sinus tachycardia at 166 bpm, hypertension of 170/100 mmHg, hypersalivation, bradypnea at 11 breaths per minute, and generalized tonic-clonic seizures that required intubation.

Laboratory analysis revealed an inflammatory syndrome (C-reactive protein: 33.5 mg/L, procalcitonin: 0.26 ng/L) with rhabdomyolysis (creatine kinase: 6079 UI/L). Toxicological screening was negative. Cerebral CT scan and angio-MRI findings were unremarkable. Electroencephalogram (The EEG was performed 10 days after the initiation of anticonvulsant treatment, which had been started prior to hospital admission) showed slow and nonreactive activity without epileptiform discharges. CSF analysis and infectious studies were normal except for a traumatic CSF sample with a white cell count of 21/mm³, 80% lymphocytes, and a red cell count of 22 000/mm³. The protein level in CSF was 0.62 g/L (normal range: 0.40–0.60 g/L), and the glucose level was 0.72 g/L. Serologic testing for HIV, HBV, HCV, CMV, EBV, HSV, VZV, TPHA, and VDRL was negative. Autoimmune conditions were suspected. However, a second cerebral MRI, antinuclear antibodies (ANA), and anti-neutrophil cytoplasmic antibodies (ANCA) were normal. Immunological testing using immunofluorescence revealed positive anti-NMDAR antibodies in both serum and CSF (Cerba laboratories) as well as low positive anti-GAD65 antibodies in serum (25.8 IU/mL, reference ≤ 17 IU/mL). Multiple glucose tests were within normal limits. Due to financial constraints, autoantibodies titers and analysis of oligoclonal bands could not be performed. However, a positron emission tomography (PET) scan for neoplasm screening was negative. Based on the 2016 autoimmune encephalitis guidelines [6], a diagnosis of anti-NMDAR encephalitis with probable anti-GAD65 encephalitis was established.

Therapeutically, the patient was treated with anticonvulsants, antipsychotics, intravenous immunoglobulins (2 g/kg), and corticosteroid pulses (15 mg/kg), followed by steroids at 1 mg/kg/day. Unfortunately, he developed septic shock due to ventilator-associated pneumonia, which was managed with appropriate antibiotics. Due to a lack of clinical improvement, he underwent five sessions of plasma exchanges, but no significant improvement was observed. According to international guidelines for the management of autoimmune encephalitis [7], Cyclophosphamide (0.6 g/m²) was initiated. However, it was later switched to rituximab (1 g) because of cyclophosphamide's potentially serious side effects, including myelosuppression, malignancy, and infertility, particularly concerning in younger patients. Subsequently, the patient showed remarkable clinical improvement, allowing for a gradual extubation on day 78 of hospitalization. Thereafter, he was transferred to another department for rehabilitation before being discharged home.

After discharge, the patient continued to exhibit mild frontal syndrome symptoms and was maintained on rituximab at a dose of 1 g every six months. At the 6-month follow-up, he had returned to his normal life without any relapses. Unfortunately, follow-up antibody titers could not be obtained due to financial constraints.

3. Discussion

Autoimmune encephalitis is the third most common cause of encephalitis, following infectious encephalitis and acute disseminated encephalomyelitis [2]. It includes a group of immune-mediated inflammatory disorders characterized by antibodies that can target different sites, such as intracellular components (e.g., Anti-GAD65), neuronal surface antigen (e.g., Anti-NMDAR), and intracellular onco-neuronal antigen (e.g., Anti-Ma) [7]. Tumor infections can trigger the production of these antibodies or can be cryptogenic [8]. This autoimmune condition can affect individuals across all age groups, with some subtypes predominantly affecting children and young adults [9].

The exact pathophysiological mechanism underlying the co-association of autoimmune encephalitis remains under investigation. However, they may involve an underlying immunogenetic predisposition and autoimmune responses triggered by infections or neoplasms. These infections can contribute to potentially autoreactive immune responses in different ways. On one hand, they may initiate or exacerbate autoimmune responses by creating a pro-inflammatory environment. Alternatively, they could disrupt peripheral tolerance mechanisms, facilitating the action of previously suppressed autoreactive effector cells [10].

On the other hand, infections may activate autoimmunity through molecular mimicry, where peptides derived from infectious agents resemble self-proteins, leading to the production of antibodies against NMDA receptors and GAD65. Nevertheless, molecular mimicry is unlikely to be the only underlying mechanism for autoimmune responses. Other mechanisms, including breaches in central tolerance, non-specific bystander activation, and persistent antigenic stimulation, may also contribute to developing autoimmune diseases [11].

Clinical manifestations are diverse and may include common symptoms such as behavioral changes, psychosis, seizures, abnormal movements, and cognitive deficits [9]. Additionally, specific symptoms are associated with particular antibodies. For instance, anti-NMDAR encephalitis is typically characterized by short-term memory impairment, orofacial dyskinesia, and autonomic dysfunction (all of which were observed in our case), whereas anti-GAD65 encephalitis may present with stiff person syndrome, limbic encephalitis, or cerebellar ataxia [12]. In our case, the patient presented only with stiff person syndrome. Other symptoms, such as limbic encephalitis and cerebellar ataxia, have been reported in only one case of this association.

Beyond clinical manifestations, autoimmune encephalitis also frequently co-occurs with other systemic or neurological conditions. For example, anti-GAD65 encephalitis is commonly linked to systemic autoimmune disease, particularly diabetes, and may co-occur with other antibodies in 19% of cases, in particular, $GABA^AR$, $GABA^BR$, and VGKC antibodies [13]. In contrast, anti-NMDAR encephalitis can be accompanied by demyelinating disorders, such as multiple sclerosis (MS), neuromyelitis optica spectrum disorder (NMOSD), and MOG antibody-associated disease (MOGAD), in 3.3% of cases [14]. Nevertheless, the coexistence of anti-NMDAR and anti-GAD65 encephalitis has been sporadically reported. The table below summarizes the main characteristics of this association based on reported cases in the literature [15, 16, 17, 18].

The median age of the reported case was 37.6 years, ranging from 18 to 66 years. There was a slight male predominance (60%), which may be related to the small sample size (n=5). Adult-onset diabetes was found in one patient (20%). A flu-like syndrome was reported as a prodromal syndrome in two patients (40%). The most common initial presentation was psychiatric symptoms, observed in three patients (60%). Seizures and abnormal movements occurred in 60% of cases, while cognitive impairment was reported in four cases

Table 1: Comparison of the profile of anti-NMDAR and anti-GAD 65 encephalitis in the reported cases

	McEntire et al [16] USA	Kammeyer et al [15] USA	Gomez Oropeza et al [17] Mexico	Calderon et al [18] Mexico	Our case
Age	58 years	66 years	18 years	28 years	18 years
Sex	F	M	F	M	M
History	Adult-onset diabetes mellitus, hypertension, bell's palsy	Chronic tobacco	NM	No history	No history
Prodromal symptoms	No	No	Headache	Covid-19	Flu like syndrome
Fever	No	No	No	Yes	Yes
Initial presentation	Cognitive decline	Neurological	Behavioral symptoms, myoclonus	Psychiatric symptoms	Behavioral and psychiatric symptoms
Psychosis	No	No	No	Yes	Yes
Seizure	Yes	No	No	Yes	Yes
Behavioral symptoms	No	Yes	Yes	Yes	Yes
Abnormal movements	No	Yes	Yes	No	Yes
Catatonia	Yes	No	No	Yes	Yes
Cognitive impairment	Yes	Yes	Yes	No	Yes
Cerebellar ataxia	No	Yes	No	No	No
Stiff person syndrome	No	No	No	No	Yes
Autonomic dysfunction	No	No	No	No	Yes
Central hypoventilation	No	Yes	No	No	Yes
Brain MRI	Normal	Limbic and brainstem encephalitis	Normal	Signal abnormalities in the bilateral anterior cingulate cortex and temporal lobes	Normal
EEG	Right temporal epileptiform discharge	Diffused and intermixed pattern	Mild generalized dysfunction	Subcortical dysfunction in the frontal, temporal and occipital regions	Slow and non-reactive activity
CSF	Pleocytosis	Pleocytosis with lymphocytic predominance	Normal	Normal	Normal
Auto-antibodies detection	NMDA: CSF GAD65: CSF and serum	NDAR: CSF GAD65: serum Ma1+Ma2: CSF	CSF	CSF	NMDA: CSF and serum GAD65: serum
Tumor	NM	Suspected lung neoplasm	NM	No	No
Treatments	GC, IVIG, RTX	IVIG, PLEX, RTX	GC, PLEX, RTX	GC, IVIG	GC, PLEX, IVIG, CYP, RTX
Time to treatment initiation	NM	2 months	NM	2 weeks	2 months
Prognosis	Favorable	Questionable outcome	Favorable	Favorable with persistent irritability and agitation	Favorable
Relapse	NM	After 1 month	After 2 months	No	No
Median Follow up	NM	1 month	4 months	6 Weeks	10 months
ICU	No	NM	No	Status epilepticus	Decreased level of consciousness
Mechanical ventilation	No	NM	No	Yes	Yes
Death	No	NM	No	No	No

F, Female; M, Male; NM, Not mentioned; CSF, cerebrospinal fluid; GC, Glucocorticoids; IVIG, Intravenous immunoglobulins; PLEX, plasma exchange; RTX, Rituximab; CYP, Cyclophosphamide; MRI, Magnetic Resonance Imaging; EEG, Electroencephalogram; NDAR, N-methyl-D-aspartate receptor; NMDA, N-methyl-D-aspartate receptor; GAD65, Glutamic acid decarboxylase, 65 kDa isoform; Ma1, Ma2, Ma1/2 antibodies; ICU, Intensive Care Unit.

(80%). Central hypoventilation was noted in two of four cases (50%).

Brain MRI findings were normal in 60% of patients (one case showed signal abnormalities, and another revealed limbic and brainstem encephalitis). EEG abnormalities were noted in 100% of the cases. CSF analysis was normal in 60% of cases. Autoantibodies testing was performed in CSF, serum or both. Neoplasm screening was conducted in 40% of cases: one result was negative, and the other revealed strong suspicion of pulmonary neoplasm (chronic

tobacco, presence of speculated pulmonary nodules with adjacent pulmonary thickening, and positivity for anti-Ma1 and anti-Ma2 antibodies).

Low titers of anti-GAD65 antibodies in serum were observed in 60% of cases, including ours. A review article by Gaspard noted that low concentrations of anti-GAD65 antibodies were associated with a heterogeneous spectrum of symptoms. None of the patients exhibited overlapping syndrome in that study, suggesting that this may be a specific future of pathogenic anti-GAD65 antibodies. The identification of GAD65 antibodies should not prevent clinicians from investigating the presence of additional antibodies, in particular, $GABA^A$, $GABA^B$ receptor antibodies, as they seem to increase the risk of an underlying neoplasm [19].

All patients received first-line immunotherapy: glucocorticoids in 80%, intravenous immunoglobulins (IVIG) in 60%, and plasmapheresis in 60% of the cases. Second-line immunotherapy with rituximab was administrated in 80% of cases, which may reflect the severity of this co-occurrence of anti-GAD65 and anti-NMDAR encephalitis. Two patients (40%) were admitted to the ICU due to neurological deterioration (status epilepticus in one case and deceased level of consciousness in the other). Relapses occurred in 40% of cases, with a median time of one and a half months. The overall prognosis was favorable in four cases, although persistent irritability and irritation were noted in one patient (20%).

The overall outcome for both types of encephalitis is generally favorable. In a cohort study of 501 patients with anti-NMDAR encephalitis by Titulaer et al., first-line immunotherapy was administrated in 95% of the patients, 57% received second-line immunotherapy, 81% had good neurological outcomes at 24 months of follow-up, 12% experienced one or more relapses, and 6% died. Additionally, 39.5% of patients had tumors, and early treatment was associated with good neurological outcome [20].

In contrast, a case series of 37 patients with anti-GAD65 neurological autoimmunity reported by Qiu et al. showed that all patients received first-line immunotherapy, 51.35% received second-line immunotherapy, and 81.3% showed a partial to good response at follow-up. However, 53.1% experienced a relapse, and 3.1% died. Tumors were suspected only in 5.4% of cases, and in this study, early treatment was not correlated with good neurological outcome [21].

In the reported cases summarized in the table below, all patients received first-line immunotherapy, 80% underwent second-line immunotherapy, 40% experienced a relapse, and only one had a tumor.

These results highlight the importance of rapid diagnosis to initiate treatment promptly and prevent prolonged ICU stays, as well as the necessity of long-term follow-up to detect early relapses and potential neoplasm. Given the small number of reported cases with co-occurring anti-NMDAR and anti-GAD65 encephalitis, further studies involving larger cohorts are needed to confirm the characteristics of this association.

4. Conclusions

The co-occurrence of anti-NMDAR and anti-GAD65 encephalitis is a rare condition that can cause severe manifestations. The nonspecific neuropsychiatric symptoms often lead to delayed or missed diagnoses. A broad antibody screening panel can prevent multiple lumbar punctures and avoid unacceptable delays in diagnosis and targeted cancer screenings.

Conflicts of Interest

The authors declare that they have no competing interests that could have influenced the objectivity or outcome of this article.

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None

Authors Contribution

All authors made substantial contributions to this case report. They were involved in patient care, the conception of the report, and the gathering of relevant clinical data. Each author participated in drafting the manuscript and revising it critically. They also reviewed and approved the final version of the manuscript, ensuring its accuracy and integrity in representing the case studied.

Data Availability

All data supporting the findings of this study are included in the article. Additional information is available from the corresponding author upon reasonable request.

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