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Case Report

A Case of Anti-NMDAR Encephalitis presenting as Acute Disseminated Encephalomyelitis (ADEM) in a Psychiatric Hospital

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Abstract

Background: Anti-NMDAR encephalitis and ADEM are different disease entities which can present with similar symptoms of neuropsychiatric disturbances, seizures, cognitive impairment, and other focal deficits. It is easy to exclude one from the other, especially for patients presenting with nonspecific symptoms.

Methods: This is the case of a 31-year-old male from the Philippines who presented with behavioral changes and refractory seizures.

Results: He was initially treated as a case of substance use disorder at our psychiatric hospital but later diagnosed with Anti-NMDAR Encephalitis and Acute Disseminated Encephalomyelitis. His CSF tested positive for both anti-NMDAR and oligoclonal bands, and his cranial MRI showed T2 Flair hyperintensities over the left cerebral hemisphere with cortical enhancement and thickening. He was treated with high-dose methylprednisolone for 5 days followed by oral steroids. His seizures stopped after steroids, and he demonstrated significant clinical improvement. His cranial MRI also showed interval resolution of the lesions.

Conclusions: ADEM and anti-NMDAR encephalitis can occur simultaneously in a patient. Even though their specific biomarkers and neuroimaging present differently, the treatment is essentially the same. Patients are treated with high-dose steroids, intravenous immunoglobulin, or plasmapheresis followed by immunomodulating therapies as warranted to prevent risk of relapse.

Keywords: Demyelinating Diseases; Anti-Nmdar; Encephalitis; Neuroimmunology; Psychiatry; Seizures

Introduction

Anti-NMDAR encephalitis is a rare, autoimmune encephalitis presenting with neuropsychiatric symptoms, seizures, movement disorders, and other focal deficits [1]. Its main pathophysiology involves autoantibodies targeting the NR1 subunit of the neuronal *N*-methyl-D-aspartate receptors or NMDAR [2]. This disease is more common in children and young adults between the ages of 15-30 years old, and is associated with good outcomes especially if adequately treated. Patients usually attain full independence (Modified Rankin Scale scores of < 2) in one to two years after treatment but some may have residual cognitive impairments [3].

On the other hand, Acute Disseminated Encephalomyelitis (ADEM) is an acute, monophasic, post-infectious illness characterized by demyelination in the brain and spinal cord. It can

develop after any preceding infection or vaccination [4]. It is more common in the pediatric population with the most frequent age of onset at around five to seven years old [5]. ADEM in adults is rare and may present more severely with worse outcomes compared to children [6]. Clinical manifestations include motor and sensory deficits, craniopathies, encephalopathy, seizures, neuropsychiatric manifestations, extrapyramidal symptoms, and signs of increased intracranial pressure [7].

The main pathology of ADEM centers on immune-mediated injury affecting the myelin sheath. The antibodies target myelin proteins such as myelin basic protein (MBP), myelin oligodendrocyte basic protein (MOBP), and myelin oligodendrocyte glycoprotein (MOG), which leads to activation of both humoral and cell-mediated immune responses.

Currently, there is no definite diagnostic criteria for both diseases. Diagnosis is still by clinical examination supported by neuroimaging and laboratory markers. For autoimmune encephalitis such as anti-NMDAR, the presence of CSF antibodies and electroencephalogram (EEG) findings of slowing, lateralized periodic discharges, or extreme delta brush can help in the diagnosis. Cranial MRI typically shows involvement of the bilateral limbic areas but other imaging patterns such as cortical/subcortical, striatal, diencephalic, and brainstem can also occur [8].

For ADEM, there is no specific laboratory biomarker that can distinguish it from the other demyelinating diseases. CSF examination may show presence of oligoclonal bands and leukocytosis. In recent years, different serum autoantibodies have been associated with ADEM such as anti-MOG, PLP (proteolipid protein), and MBP [9]. On neuroimaging, there is presence of T2-weighted hyperintense asymmetrical white matter lesions on cranial MRI and longitudinally-extensive lesions extending over multiple segments in spine MRI [10].

Although with different pathogenesis, treatment of both anti-NMDAR encephalitis and ADEM centers around the same immune modulation therapy. High-dose methylprednisolone, intravenous immunoglobulin (IVIg), and plasmapheresis are all possible modes of therapy. Supportive treatment such as airway protection for those with altered sensorium, antiseizure medications, and antipsychotics for neuropsychiatric symptoms may be given. Some patients may also need long-term corticosteroids or immunomodulators such as Rituximab or Cyclophosphamide, especially those with relapsing episodes [11].

For anti-NMDAR encephalitis, some patients have concomitant ovarian teratomas or testicular tumors so removal of the tumor is also warranted for complete recovery. Rehabilitation therapy and serial monitoring of patients especially those with residual deficits are required for further care.

Case Report

We now have the case of a 31-year-old male from the Philippines who is a known alcoholic for 14 years with almost daily intake of lambanog (palm liquor). Three days prior to consultation, he developed multiple generalized tonic clonic seizures. He also started having behavioral changes like shouting randomly, repeatedly talking to himself, and answering inappropriate responses. Due to the changes in his behavior, he was brought to our psychiatric hospital, and was assessed as a case of Substance Use Disorder (Ethanol). He was given Diazepam and Vitamin B Complex at the emergency room.

Upon admission, the seizures resolved, but the patient still had psychiatric symptoms so he was started on antipsychotics. Various laboratory exams were requested which were all normal except for mild serum hyponatremia. A Cranial CT scan was done which showed diffuse cerebral edema. This was initially attributed to the patient's hyponatremia. He was then started on low-dose Mannitol, and advised hydration and cautious correction of sodium.

Two weeks from admission, he started having focal seizures again with head version to the right and clonic movements of the right arm and right leg, accompanied with post-ictal aphasia. He would experience two to three focal seizures per hour with no loss of consciousness in between. He was then treated as a case of *epilepsia partialis continua* and was started on 3gm of Levetiracetam and 300mg of Phenytoin divided in 3 doses. Despite these medications, the seizures still persisted and he was eventually hooked to Midazolam drip for better seizure control.

Due to his intractable focal seizures, a repeat Cranial CT with Contrast was requested which now showed edema mainly on the left cerebral hemisphere with cortical enhancement (Figure 1).

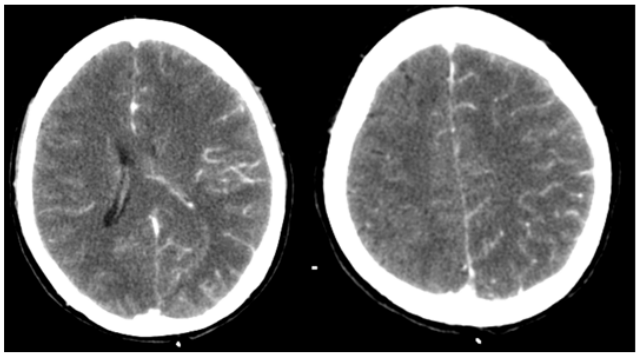

Figure 1: Cranial CT with contrast showing cortical enhancement of the left cerebral hemisphere.

The initial diagnosis based on the CT scan was left cerebritis, probably infectious in origin. A lumbar puncture was requested, and he was empirically started on meningitic doses of Acyclovir and Ceftriaxone to cover for both viral and bacterial meningitis. Dexamethasone was also given for the edema. Full workup including complete blood count, metabolic panel, ESR, CRP, procalcitonin, STD panel, and HIV test were done which all came back negative.

His CSF results showed mononuclear lymphocytosis with negative bacterial, viral, and fungal CSF panels, normal cell cytology, and negative CSF cultures. A 21-channel routine electroencephalogram (EEG) was also done which showed unequivocal focal slowing in the left cerebral hemisphere.

However, even with the antiseizure medications and antibiotics, his seizures still persisted and Perampanel 2mg daily was added. Patient was also still aphasic. Because he did not respond to his previous medications, we now entertained the possibility that this was not infectious in nature. We thought of other causes of encephalitis or other structural lesions in the left hemisphere (such as an intracranial mass) causing persistent focal seizures, so a cranial MRI was requested (Figure 2).

With the Cranial MRI showing cortical hyperintensities, we now thought of possible demyelinating diseases such as Acute Disseminated Encephalomyelitis (ADEM) or MOG Antibody-Associated Disease (MOGAD). The patient was cleared by Infectious Disease service and was immediately started on Methylprednisolone pulse therapy (MPPT) with a dose of 1 gm/day for five days. After

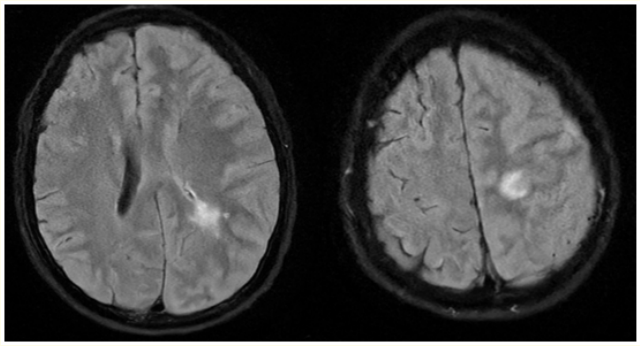

Figure 2: Cranial MRI showed T2/Flair hyperintensities along the left frontal and parietal lobe sulci. There are also hyperintense lesions at the juxtacortical region of the left precentral gyrus with minimal restricted diffusion and non-enhancement. The left cerebral sulci and fissures are effaced with compression of the left lateral ventricle. There is also noted cortical thickening at the left hemisphere.

2 days of MPPT, his seizures suddenly stopped and there was significant improvement with the patient's sensorium. MPPT was continued for three more days and the Cranial MRI was repeated post-steroid therapy (Figure 3 and Figure 4). The patient's serum MOG came back negative but his CSF tested positive for oligoclonal bands. Even though the presence of oligoclonal bands in the CSF is not specific for ADEM, this gave stronger evidence that this was due to a demyelinating disease. A spine MRI was included in the diagnostic workup; however, resource limitations at the hospital delayed the examination until two months after the MPPT, which yielded normal results.

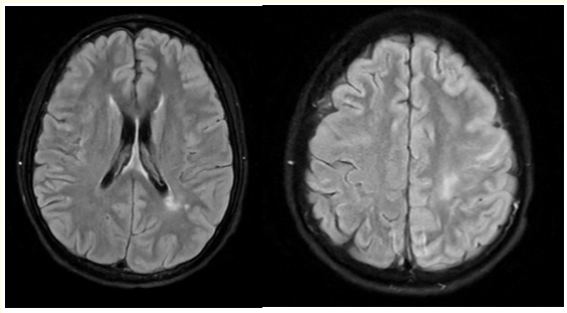

Figure 3: The Cranial MRI post-MPPT now showed interval resolution of the T2-flair hyperintensities along the left frontal and parietal lobe sulci. There are still some residual hyperintense lesions at the left precentral gyrus but the cerebral edema is also resolving.

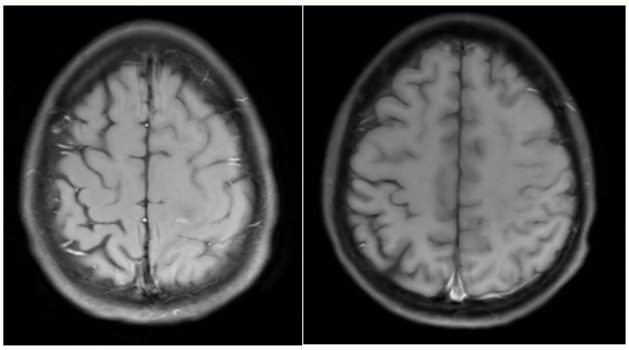

Figure 4: There is still residual cortical thickening over the left cerebral hemisphere but already improved compared to the cranial MRI pre-MPPT.

We were ready to conclude the case as ADEM when the CSF anti-NMDAR antibody results became available and were also positive. This raised the possibility of overlapping ADEM and anti-NMDAR autoimmune encephalitis. Consequently, a scrotal ultrasound was performed to evaluate for an underlying neoplasm, but the findings were unremarkable

The patient was discharged stable with a Modified Rankin Scale (MRS) score of 1. There was no recurrence of seizures, and he was discharged on a single antiseizure medication. His psychotic symptoms had completely resolved, with only mild word-finding difficulty at the time of discharge. He was continued on oral corticosteroid therapy to reduce the risk of relapse associated with anti-NMDAR encephalitis, with the dose gradually tapered before eventual discontinuation.

Discussion

Overlapping syndromes involving anti-NMDAR encephalitis and demyelinating diseases are uncommon. Patients may present with clinical features of both and it is important to recognize these cases properly as their prognosis may differ. While patients with anti-NMDAR encephalitis usually respond well to immunotherapy, the presence of concomitant demyelinating diseases such as ADEM may require a more aggressive or longer treatment approach [12]. Approximately a quarter of ADEM patients subsequently develop multiple sclerosis, so serial neuroimaging is important to monitor disease progression [13]. In our patient, both a cranial and a spinal MRI were done to serve as baseline imaging.

Relapses occur in up to 20% of patients within two years of the initial diagnosis [14]. Factors that can contribute include inadequate treatment, early steroid withdrawal, or just intrinsically aggressive progression of the disease [15]. Although some studies recommend tapering steroids over 4-6 weeks [16]. our patient underwent a more gradual taper over 12 weeks to minimize relapses. Regardless of the tapering duration used, close clinical and radiologic follow-ups remain essential to detect new focal deficits or recurrent disease activity.

In contrast, anti-NMDAR encephalitis is known be associated with underlying neoplasms, more commonly ovarian teratomas and testicular tumors. Early detection and surgical removal of tumors, when present, have been associated with improved clinical outcomes and a higher likelihood of complete symptom resolution [17,18]. Our patient has normal scrotal ultrasound findings which excludes an underlying tumor as a potential trigger for the encephalitis.

This case highlights the importance of recognizing overlapping neurologic and psychiatric manifestations. The patient was initially managed as a case of substance use disorder because of his history of chronic alcohol use and new-onset psychosis. However, the subsequent development of refractory focal seizures and the lack of clinical improvement despite antimicrobial therapy and multiple antiseizure medications, prompted further investigation and consideration of alternative diagnoses.

This case is notable for the occurrence of both anti-NMDAR encephalitis and acute disseminated encephalomyelitis. Although such overlap syndromes are uncommon, it is important that we are aware that these conditions can occur together because this can guide the diagnostic approach and workup.

There are still no definite diagnostic criteria for both diseases. Diagnosis relies on a combination of clinical, radiologic, and laboratory findings. Anti-NMDAR encephalitis usually has a normal MRI while ADEM appears as asymmetrical, multifocal T2 hyperintensities on cranial MRI and longitudinally extensive lesions on spinal MRI. In our patient, his cranial MRI findings resemble ADEM more with its preferential involvement of the juxtacortical regions in the left cerebral hemisphere. However, his CSF tested positive for anti-NMDAR antibody, raising the possibility of concurrent anti-NMDAR encephalitis.

The marked radiological and clinical resolution of the patient's symptoms after MPPT provided additional support for the diagnosis of an ADEM and anti-NMDAR encephalitis overlap syndrome [19]. Even though the diagnosis was delayed in this case, the patient still had good outcomes owing to the high-dose steroid therapy, followed by a gradual tapering of his oral steroid. Overall, this case was a diagnostic dilemma initially but proved to be a good learning case in our psychiatric hospital to look beyond psychiatric manifestations and consider all possible diagnoses.

Conclusion

We present the case of an adult Filipino male who developed refractory seizures and behavioral changes and was subsequently diagnosed with both anti-NMDAR encephalitis and acute disseminated encephalomyelitis (ADEM). These conditions may share overlapping clinical manifestations but can be distinguished by their characteristic neuroimaging findings and laboratory biomarkers. This case highlights the importance of recognizing that anti-NMDAR encephalitis and ADEM can occur together and may initially mimic primary psychiatric disorders, which can cause a delay in diagnosis. Although both conditions are treated with immunotherapy, prompt identification of each disease entity is important for diagnostic evaluation, prognostication, and long-term monitoring.

Disclosure

The authors have nothing to disclose. No conflict of interest.

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Informed Consent

Informed consent was obtained from the patient included in the research.

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