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Case Report

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Possible Autoantibody-Negative Autoimmune Encephalitis in a Sixty-Four-Year-Old Man Post-Varicella-Zoster Virus Vaccination

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Abstract

Autoimmune encephalitis (AIE) is a rare condition of brain inflammation that can be the result of multiple etiologies such as neoplasms of the ovaries, teratomas, and, more rarely, post-vaccine administration. Vaccine-associated AIE has been reported and described in the literature with vaccines against yellow fever, tetanus, diphtheria, pertussis and polio, Japanese encephalitis, coronavirus disease 2019 (COVID-19) mRNA, and ChAdOx1 nCoV-19 vaccine. Additionally, there are few reported cases of AIE secondary to varicella-zoster vaccine administration. However, these numbers are found in large population studies and none of the cases have been further described. AIE has a variable presentation with non-specific prodromal symptoms occurring in the early stages with progression to neuropsychiatric and dysautonomia in the late stages. Therefore, this presentation introduces diagnostic difficulty especially in the absence of significant laboratory findings. Here, we discuss all reported and described cases within the literature on vaccine-associated AIE and their respective presentations. We also report and describe a case of possible AIE in a 64-year-old male, 1 week post-varicella-zoster vaccine administration. We further discuss the epidemiology, differential diagnosis, treatment, and prognosis of AIE as well as when to raise clinical suspicion for AIE associated with vaccine administration.

Keywords: Varicella-zoster vaccine; Post-vaccination encephalopathy; Autoimmune encephalitis; Autoimmune; Encephalitis; NMDA receptor encephalitis; Catatonia; Antibody negative

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Introduction

Autoimmune encephalitis (AIE) is a rare, immune-mediated condition involving autoantibody production leading to brain inflammation. The condition serves as an alternative diagnosis when infectious causes do not explain neurological symptomatology [1]. The prevalence of AIE as of January 2014 in a population-based study was 13.7/100,000, and its demonstrated incidence between 1995 and 2015 was 0.8/100,000 [2]. Anti-N-methyl-D-aspartate receptor (NMDAR) encephalitis is one of many subtypes of AIE and is the most well-studied. It was first described in women presenting with newfound psychiatric, neurological, and autonomic symptoms in the setting of ovarian teratoma [3]. While paraneoplastic syndrome-related AIE continues to be studied, other etiologies, such as post-vaccine administration AIE, have been reported. The diphtheria, tetanus, and acellular pertussis vaccine (DTaP), as well as the inactivated polio vaccine (IPV), yellow fever vaccine, and human papillomavirus (HPV) vaccine, have been found to have the highest reporting odds ratios for AIE [4]. The varicella-zoster virus (VZV) vaccine has increased in use over time, with 34.5% of adults aged 60 and over receiving the vaccine in 2018 compared to 6.7% in 2008 [5]. Although five cases of VZV vaccine-associated AIE have been reported in the literature, none have yet to be described [4]. This increasing trend in administration of the VZV should heighten vigilance for vaccine-associated side effects such as constitutional symptoms, reactivation of latent virus, and rare conditions such as AIE.

Classic features of AIE are broad but begin after a non-specific prodromal phase followed by psychiatric symptoms, including irritability, aggression, emotional lability, hallucinations, catatonia, and marked disturbances of the sleep-wake cycle. Memory deficits and speech abnormalities are also commonly seen. The psychiatric phase persists for approximately 1 month before movement disorders manifest in the form of tremors, orofacial dyskinesias, and truncal/extremity hyperkinesia. General or focal seizures that may result in status epilepticus can occur at any point during the condition but appear

Table 1. Lumbar Puncture Findings of Patient During Hospital Stay

Laboratory value	Day 1	Day 4	Day 10	Reference ranges
White blood cell count	1 cell/µL	0 cells/µL	2 cells/µL	0 - 5 cells/µL
Lymphocyte percentage	89%	-	60%	40-80%
Protein	90 mg/dL	42 mg/dL	55 mg/dL	15 - 45 mg/dL
Glucose	72 mg/dL	54 mg/dL	73 mg/dL	50 - 80 mg/dL

to occur more frequently in the male sex. Other symptoms can include dysautonomia with tachy-/bradycardia, hypo-/hypertension, hypo-/hyperthermia, as well as hypo-/hyperventilation [3]. Due to the constellation of features and nonspecific laboratory and imaging findings in AIE, patients are commonly misdiagnosed with a psychiatric disorder. Additionally, as the condition commonly presents in young patients, AIE is often challenging to diagnose in individuals above the age of 50.

In this case report, we discuss the etiologies, diagnostic difficulties, laboratory and imaging findings of AIE, and one rare cause of the disorder. Here, we present a case, not previously described in the literature, of possible AIE in a 64-year-old male with no previous psychiatric history and a past medical history of essential hypertension and cardiomyopathy 1 week after being administered a VZV vaccination.

Case Report

A 64-year-old man with a past medical history of unspecified cardiomyopathy and essential hypertension presented to the emergency department (ED) for 3 weeks of insomnia, memory loss, bizarre statements, blepharospasm, chest pain, paresthesias, and seizure-like activity. The patient had no previous psychiatric history, risk factors, or history of psychiatric medications. Of note, 1 week prior to the onset of these symptoms, the patient received a VZV vaccination. Near the onset of these symptoms, he was evaluated twice at the ED prior to hospital admission. Workup at that time included a computed tomography (CT) of head, computed tomography angiography (CTA) of chest, and cardiac stress test. Imaging and all other appropriate labs were negative, and the patient was discharged for follow-up with neurology and his primary care physician (PCP).

At his PCP, the patient was initially prescribed lorazepam for anxiety, which he stopped due to lightheadedness, and then was subsequently initiated on sertraline and quetiapine for continued anxiety and insomnia. His neurologist ordered an outpatient magnetic resonance imaging (MRI) of the brain, MRI of the cervical spine, electroencephalogram (EEG), and carotid ultrasound, but he was unable to complete these due to subsequent hospital admission. At his home, the patient's family found him unresponsive, with his eyes open, blood in his mouth, and tremors in his right upper extremity, which generalized to a full tonic-clonic seizure. The patient was then transported by emergency medical services, during which a second seizure episode occurred and was controlled with intravenous (IV) lorazepam.

On initial exam in the ED, the patient was noted to be postictal. His neurological exam revealed no focal deficits and no myoclonus. The patient was able to follow simple commands and had minimal, broken speech. Workup began with concern for meningitis. Lumbar puncture (LP) in the ED was remarkable for elevated cerebrospinal fluid (CSF) protein of 90 mg/dL with 89% lymphocyte predominance (Table 1). The patient was then started on 750 mg levetiracetam twice daily, admitted, and neurology was consulted. After admission, the patient began experiencing urinary incontinence, active hallucinations, and delusions, yet on questioning, he was fully oriented, and able to follow commands. His neurological exams on admission repeatedly demonstrated no focal deficits with intact cranial nerves, intact coordination, sensation grossly intact to light touch, motor exam with no upper or lower extremity drift and intact strength, and 2+ deep tendon reflexes throughout the upper and lower extremities. An MRI of the brain without contrast was performed at this time, which was unremarkable. Two days after admission, the patient demonstrated altered mental status, with disorientation and bizarre speech that progressed to being nonverbal. On neurological examination, the patient was largely non-participatory but would intermittently attend to the examiner with purposeful movements only in the right upper extremity. Subsequent exams showed waxing and waning mental status as the patient was intermittently nonverbal and hyperverbal. The patient was then placed under continuous video EEG (vEEG) monitoring for 120 h. The vEEG showed generalized irregular delta/theta activity with no electrographic seizures or interictal epileptiform discharges. These results were consistent with mild generalized encephalopathy. Periods of agitation, right arm shaking, and eyelid fluttering caught on vEEG were without EEG correlate.

Due to high suspicion of AIE, the patient was started on 30 g intravenous immunoglobulin (IVIG) and methylprednisolone. Repeat LP and MRI of the brain with and without contrast were performed, which returned unremarkable. A few days later, the patient became catatonic, increasingly agitated, and paranoid. Psychiatry was consulted, and their recommended treatment of 4 mg IV midazolam and 200 mg quetiapine demonstrated minimal improvement. A third LP was performed, which showed an elevated protein of 55 mg/dL. High-dose prednisone was initiated but was discontinued after ENC-2/onconeural antibody panel results were negative at this time. Plasmapheresis (PLEX) was then attempted since the patient demonstrated inconsistent improvement with IVIG, methylprednisolone, or prednisone. Symptoms markedly improved on PLEX, which continued throughout the hospitalization, and prednisone was restarted. The patient was then discharged.

After returning home, his family noted restoration of baseline behavior, with a lack of paranoia, hallucinations, and delusions, with a return to normal activities of daily living. Se-

quelae included continued dizziness, fatigue, and intermittent tremors, for which lorazepam, quetiapine, and clonidine were prescribed. These symptoms have gradually improved over the next year post-discharge, but the patient continues to struggle with an action tremor that interferes with fine motor activities.

Discussion

Here, we present a case of a 64-year-old male with no psychiatric history who subsequently developed possible AIE post-VZV vaccination. As reported in the literature, AIE is rare, predominantly occurring in young females with a reported incidence of 0.8/100,000 [2, 6]. Vaccination-associated AIE is even more rare, with only 11 cases published in the literature (Table 2 [7-16]), with this being the first case thoroughly described post-VZV vaccine.

In our case, the differential diagnoses of meningitis, septic encephalopathy, neoplasm, cerebrovascular disease, epileptic disorders, metabolic disorders, drug toxicity and withdrawal, psychiatric disorders, and functional neurological disorders were excluded. Prior to admission, this patient was an otherwise healthy middle-aged man with no history of neuropsychiatric illness or autoimmune disease, who developed sub-acute onset encephalopathy post-VZV vaccine with subsequent improvement on PLEX, IVIG, and corticosteroid treatment. Initial brain MRI, including T1, T2, fluid-attenuated inversion recovery (FLAIR), and diffusion-weighted, showed no discrete mass, mass effect, hemorrhage, infarction, or other pathological variation in signal. A repeat MRI on the third day of admission was similarly unremarkable. CT of the chest, abdomen, and pelvis was performed to assess for possible neoplasm; however, these studies were negative. A pelvic and scrotal ultrasound was not pursued because the CT revealed no abnormalities of the pelvic region or genitalia. Initial LP was remarkable for elevated CSF protein of 90 mg/dL with 89% lymphocyte predominance (Table 1). Meningitis/encephalitis panel for bacterial, fungal, and viral (including herpes simplex virus type 1 (HSV1), HSV2, and VZV) pathology was negative at 24 and 72 h post-admission. Given the patient's overall symptoms, imaging and laboratory studies, and improvement with PLEX, IVIG, and corticosteroids, the presumptive diagnosis of AIE was made.

The diagnostic criteria, or "Graus" criteria for AIE, are well documented in the literature and categorized into possible, probable, and definite [17]. The gold standard for confirmation of AIE is the presence of autoantibodies in the serum and/or CSF with increased sensitivity and specificity if both laboratory tests return positive. Our current understanding is that the absence of autoantibodies does not exclude AIE as antibody testing may be negative in up to 50% of all AIE cases [17, 18]. Although our patient fits the criteria for possible AIE with a sub-acute presentation of 3 weeks, short-term memory loss, psychiatric symptoms, seizures, lymphocytic predominance in CSF samples, and exclusion of other alternative diagnoses, definite AIE criteria were not met due to the absence of autoantibodies in the patient's serum and CSF [17]. Furthermore, the criteria for probable anti-NMDAR encephalitis were also met with abnormal psychiatric behavior including catatonia, seizures, autonomic dysfunction, lymphocytic predominant CSF, and exclusion of alternative diagnoses [17]. During hospitalization, these symptoms raised a high suspicion for AIE that was missed on the onconeural panel, which prompted treatment to delay progression and possible sequelae. Although subsequent serum/CSF autoantibody testing, brain MRI, or brain biopsy could have been pursued to determine a definitive diagnosis of AIE, further invasive workup was not considered given the patient's symptom resolution with PLEX treatment.

Catatonic symptoms of immobility, staring, mutism, and posturing are the most frequent manifestations observed and can be used to distinguish AIE from other psychotic presentations. Catatonia is a predominant symptom in 70.6% of patients with anti-NMDAR encephalitis [19]. The psychiatric manifestations of AIE have become so prominent that many psychologists now include AIE as a differential in patients presenting with psychosis with no history of psychotic disorders [20].

Treatment in AIE targets the underlying etiology and symptoms of encephalitis. First-line therapy includes immunotherapy, corticosteroids, IVIG, and PLEX. If there is an associated teratoma, as commonly seen with female patients, surgical removal is first line with treatment of associated symptoms. Given the high occurrence of behavioral manifestations, sedation is often considered for PLEX. Second-line treatment of AIE includes immunosuppressors such as rituximab or cyclophosphamide, often prioritized in cases of late diagnosis [21]. However, appropriate treatment in most cases is usually delayed due to the condition being misdiagnosed as psychiatric or other disorder. In our patient, IVIG was initiated, followed by methylprednisolone. Treatment with IV midazolam and quetiapine demonstrated inconsistent improvement. AIE symptoms markedly improved for our patient on PLEX.

The prognosis differs between teratoma-associated AIE, and AIE alone, due to the potential for teratoma relapse and surgical removal of the tumor. Most patients with AIE experience symptom resolution within 4 weeks of treatment with immunosuppression when there is no delay in treatment, with 97% having a good outcome and 12% having clinical episodic relapse by the 2-year mark [22]. As shown in Table 2, spontaneous remission is possible with only conservative therapies. In the cases of misdiagnosis or delayed treatment, patients are at risk of experiencing complications, including cognitive impairment, sleep disorders, hyperkinesia, autonomic dysfunction, persistent amnesia, coma, and status epilepticus [1]. In the 2 years since discharge from the hospital, our patient continues to experience residual action and postural tremors with episodes of exacerbation which highlights the importance of early diagnosis and treatment of AIE.

Our case highlights two unique points among the vaccine-associated AIE literature. 1) In a patient with usual AIE symptomatology and recent vaccine administration, appropriate treatment should be considered and initiated early to prevent potential sequelae. 2) When compared to other reported cases of vaccine-associated AIE, our patient had a notably prolonged course of symptoms with long-lasting sequelae.

As discussed in the prognosis of patients with AIE from the literature, early identification and treatment are necessary to stop

Table 2. Clinical Cases of Autoimmune Encephalitis Following Vaccination

Case	Sex	Age	Etiology	Time until onset	Symptoms	Positive lab and imaging findings	Effective treatment and course
Coecklebergh and Reynders, 2021 [7]	M	29	Yellow fever vaccine	2 to 3 days post-vaccination	Systemic, neurological, movement, autonomic	Anti-NMDA antibodies in serum and CSF. LP: WBC 1 cell/µL, protein 54 mg/dL	Spontaneous remission after 3 weeks with no intervention
Hozakova et al, 2018 [8]	F	17	Yellow fever vaccine	27 days post-vaccination	Systemic, neurological, psychiatric	Anti-NMDAR antibodies in serum and CSF. EEG: slow wave delta activity.	-
Hofmann et al, 2011 [9]	F	15	Tetanus diphtheria pertussis and polio vaccine (TdaP-IPV)	1 day post-vaccination	Systemic, psychiatric, movement, neurological, autonomic	Anti-NMDAR antibodies in serum and CSF	-
Wang, 2017 [10]	F	2	Japanese encephalitis vaccine	14 days post-second dose of JE vaccination	Systemic, psychiatric, neurological, movement	Anti-NMDAR antibodies in CSF	IVIG
Abu-Abaa et al, 2022 [11]	M	75	mRNA COVID-19 vaccine	1 day post-vaccination	Neurological, psychiatric	LP: WBC 9 cells/µL, 62% lymphocytes, protein 76 mg/dL, glucose 75 mg/dL. Myelin basic protein 29.6 ng/mL.	Complete resolution over 5-day hospitalization without active intervention.
Sluyts et al, 2022 [12]	M	48	mRNA COVID-19 vaccine	6 days post-vaccination	Psychiatric, neurological	LP: WBC 34 cells/µL, 95% lymphocytes, protein 107 mg/dL.	Complete resolution over 1 week of hospitalization without active intervention.
Peraita-Adrados and Bravo-Quelle, 2024 [13]	M	23	mRNA COVID-19 vaccine	7 weeks post-vaccination	Systemic, neurological, psychiatric, autonomic	Positive SARS-CoV-2 antigen test. LP: highly elevated protein and lymphocytic pleocytosis. Diffusion weighted MRI showed high signal in left hemisphere involving cerebral cortex, temporal, and frontal lobes.	Six cycles of PLEX with daily 1 g corticosteroid, followed by steroid taper.
Takata et al, 2021 [14]	F	22	ChAdOx1 nCoV-19 vaccine	A few days post-vaccination	Systemic, neurological, psychiatric	LP: WBC 18 cells/µL, 100% lymphocytes, IgG oligoclonal bands.	Olanzapine, with continued maintenance dose and follow-up as she has not fully recovered post-discharge.
Zuhorn et al, 2021 [15]	F	21	ChAdOx1 nCoV-19 vaccine	5 days post-vaccination	Neurological	LP: WBC 46 cells/µL. EEG: diffuse abnormally slow theta rhythms.	10 mg dexamethasone with significant improvement.
Zuhorn et al, 2021 [15]	F	63	ChAdOx1 nCoV-19 vaccine	6 days post-vaccination	Neurological, psychiatric, movement	LP: WBC 115 cells/µL. EEG: diffuse abnormally slow theta rhythms.	5 g methylprednisolone over 5 days led to immediate improvement.
Zuhorn et al, 2021 [15]	M	63	ChAdOx1 nCoV-19 vaccine	8 days post-vaccination	Systemic, neurological	LP: WBC 7 cells/µL.	Steroid immunotherapy rejected by patient, complete resolution over several weeks.

All 11 cases of vaccine-associated AIE in the literature and their associated characteristics are reported and described. Of note, other cases have been reported, but not described within the literature [8, 16]. AIE: autoimmune encephalitis; COVID-19: coronavirus disease 2019; CSF: cerebrospinal fluid; EEG: electroencephalogram; IVIG: intravenous immunoglobulin; LP: lumbar puncture; NMDAR: N-methyl-D-aspartate receptor; PLEX: plasmapheresis; SARS-CoV-2: severe acute respiratory syndrome coronavirus 2; WBC: white blood cell.

the neurological sequelae of the disorder. As seen in our patient, with a prolonged hospital stay and sequelae, delayed intervention may have permanent neurological consequences. Patients with usual AIE symptoms who present with a history of recent vaccine administration should be considered for diagnostic testing and treatment earlier than patients with usual AIE symptoms without a history of recent vaccine administration.

Of the 11 other cases reported in Table 2, all patients' symptoms resolved within weeks of diagnosis with minimal sequelae [7-16]. Our patient had a prolonged hospital course of 35 days, with noted clinical improvement only after IVIG, methylprednisolone, and PLEX were initiated. The difference between our case and the 11 other vaccine-associated AIE cases reported in the literature is the live attenuated VZV vaccination used. Since the VZV vaccine is live attenuated, the antigenicity of this vaccine is higher relative to the other vaccines used, which may have triggered a heightened immune response in our patient, leading to more autoantibodies produced and, therefore, prolonged symptoms and sequelae.

Learning points

Our patient represents the first described case of possible AIE associated with Varicella-Zoster vaccination. In patients presenting with non-specific prodromal symptoms followed by psychiatric/neurological manifestations in the peri-vaccine period, clinicians should have heightened suspicion for AIE. For cases in which there is high suspicion, treatment should be given promptly as a delay could result in prolonged hospital courses and permanent neurological sequelae. Prompt treatment is also important as AIE commonly can be seronegative at the time of diagnosis.

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Conflict of Interest

No competing financial interests exist. None of the authors stated above have any conflict of interest. This manuscript does not report on any clinical trials.

Informed Consent

Informed consent was obtained from the patient during their hospital stay.

Author Contributions

John Santare: analysis of data, drafting of manuscript, drafting of figures, review and editing; Robert Murphy: analysis of data, drafting of manuscript, drafting of figures, review and editing; Yaniv Maddahi: drafting of manuscript, review and editing; Sydnee Goyer: drafting of manuscript, review and editing; Harsh Bhalala: drafting of manuscript, review and editing; Barbara Solis: analysis of data, review and editing; Ilya Bragin: review and editing; Sameer Ali: review and editing; Mina Aiad: conception and design of study, acquisition of data, review and editing.

Data Availability

The authors declare that data supporting the findings of this study are available within the article.

Abbreviations

AIE: autoimmune encephalitis; CSF: cerebrospinal fluid; CT: computed tomography; ED: emergency department; EEG: electroencephalogram; IVIG: intravenous immunoglobulin; LP: lumbar puncture; MRI: magnetic resonance imaging; NMDA: N-methyl-D-aspartate; PCP: primary care physician; PLEX: plasmapheresis; vEEG: video electroencephalogram; VZV: varicella-zoster virus

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