CASE RECORDS of the MASSACHUSETTS GENERAL HOSPITAL

Case 22-2025: A 19-Year-Old Woman with Seizurelike Activity and Odd Behaviors

Judith A. Restrepo, M.D.,[1,2] Amirkasra Mojtahed, M.D.,[3,4] Leah W. Morelli, M.D.,[1,2] Nagagopal Venna, M.D.,[5,6] and Gulisa Turashvili, M.D., Ph.D.[7,8]

PRESENTATION OF CASE

Author affiliations are listed at the end of the article.

N Engl J Med 2025;393:488-96.
DOI: 10.1056/NEJMcpc2412531

CME

Dr. Jasmine N. Jack (Psychiatry): A 19-year-old woman was admitted to this hospital because of episodes of shaking of the right arm and leg and odd behaviors.

The patient had been in her usual state of health until 10 days before the current presentation, when slowed speech developed, along with intermittent shaking and numbness of the right arm. Seven days before the current presentation, bystanders witnessed the patient collapse while she was standing on a subway platform; full-body shaking reportedly occurred. On arrival of emergency medical services, the patient was confused, drooling, and had bitten her tongue. During transport to the emergency department of another hospital by ambulance, she gradually became more alert.

In the emergency department, the patient did not recall the events at the subway station. The vital signs and physical examination were normal. The blood lactate level was 13.9 mmol per liter (125.2 mg per deciliter; reference range, 0.7 to 2.1 mmol per liter [6.3 to 18.9 mg per deciliter]), and the blood creatine kinase level was 84 U per liter (reference range, 30 to 135). Blood levels of electrolytes, glucose, alanine aminotransferase, and aspartate aminotransferase were normal, as were the results of tests of kidney function and the complete blood count. Computed tomography (CT) and magnetic resonance imaging (MRI) of the head, performed without the administration of intravenous contrast material, showed no abnormalities. The patient was admitted to the other hospital.

On the first hospital day, the patient had three episodes of sudden, intense fear and dread. Each episode was followed by full-body shaking that was witnessed by the nurse and lasted approximately 60 to 90 seconds. During the third episode, she had apnea, and the oxygen saturation decreased to 50% while the patient was breathing ambient air. Supplemental oxygen was delivered through a bag–valve–mask device, and treatment with intravenous lorazepam and levetiracetam was administered.

On the second hospital day, treatment with lamotrigine was started. The patient had several episodes of stuttering speech, and treatment with daily oral levetiracetam

was started. On the third hospital day, electroencephalography (EEG) showed no evidence of electrographic seizures or epileptiform patterns. On the fifth hospital day, she was discharged home with plans to continue treatment with lamotrigine and levetiracetam and follow up in a neurology clinic once she returned to the state where she was attending college.

On the way home from the hospital, the patient's parents noted rhythmic movements of the patient's mouth and shaking of the right arm. When they spoke to her, she stared and could not respond. They took her back to the emergency department of the other hospital.

On evaluation, the patient could not speak; however, she communicated by typing messages on her mobile phone. During the examination, an episode of twitching of the face and jaw occurred, but the remainder of the neurologic examination was normal. The blood level of lactate was normal. The patient was readmitted to the other hospital.

During the subsequent 2 days, the patient had intermittent episodes of mutism and twitching of the right arm and leg. An EEG was again reportedly normal. On the third hospital day, the patient was discharged home.

One day after discharge from the other hospital, the patient had persistent numbness of the right arm, twitching of the right arm and leg, and intermittent mutism. The parents brought her to the emergency department of this hospital.

Additional history was obtained from the patient's parents. She had a history of depression and anxiety symptoms, for which she had seen a therapist briefly in the year before the current presentation; there were no previous psychiatric admissions. Medications since discharge from the other hospital included levetiracetam, lamotrigine, melatonin, and folic acid; she had been taking no medications before the admission to the other hospital. She had no known adverse reactions to medications. The patient was born and raised in an urban area of South Asia. One year before the current presentation, she moved to a suburban area of the Midwest United States, where she attended college and had two part-time jobs. Three weeks before the current presentation, she traveled with her parents to New England for vacation. The patient did not drink alcohol, smoke cigarettes, vape, use cannabis, or take illicit drugs. Her mother, father, and sibling were alive and well; her paternal grandmother had schizophrenia.

On examination, the temporal temperature was 36.3°C, the blood pressure 109/62 mm Hg, and the pulse 82 beats per minute. The patient was alert, oriented, and appeared comfortable. She was lying in bed and unable to sit up on request; later, during the examination, she was able to sit up spontaneously. At times, she spoke fluently in English, and sometimes she spoke in her native language even when an interpreter was not present. At other times, she was mute, or speaking was effortful, and she paused after every one to two words or said half a sentence repeatedly while trying to ask a question. It was again noted that she was able to respond intermittently by typing on her mobile phone. The thought process was sometimes incoherent and sometimes organized and goal-directed. She was able to say the months of the year backward rapidly and accurately. There was no pronator drift. Intermittent nonrhythmic jerking of the right arm and leg was observed approximately every 10 to 60 seconds. The strength was normal in both arms and in the left leg. She was unable to lift the right leg when asked; however, Hoover's sign was present, a finding suggestive of nonparesis of the right leg. When the feet were tickled, both legs lifted rapidly. Sensation and deep-tendon reflexes were normal. The gait could not be assessed because the patient described being unable to walk. The blood level of lactate was 3.4 mmol per liter (30.6 mg per deciliter), and the blood level of creatine kinase was 890 U per liter. Blood levels of electrolytes, glucose, alanine aminotransferase, and aspartate aminotransferase were normal, as were the results of tests of kidney function and complete blood count. The patient was evaluated by the neurology consultant. Treatment with antiepileptic medications was continued, and she was admitted to this hospital.

On the second hospital day, the patient was evaluated by the psychiatry consultant. During the interview, she had a sudden onset of extension and stiffening of the right arm, flexion and stiffening of the left arm, and turning of the head to the right, followed by full-body shaking. During the episode, the eyes stared but she blinked in response to threat. After approximately 60 seconds, the episode resolved. She described feeling that her "brain and mind were disconnected," and she did not think that she would be

able to return to college. She did not have auditory or visual hallucinations or suicidal or homicidal thoughts.

Later that evening, the patient received treatment with oral lorazepam because of anxiety, and she subsequently became agitated and tried to run out of the hospital room. On evaluation, she did not make eye contact, follow directions, or respond to questions. She pressed the call button repeatedly even when staff were in the room. She said both sensical and nonsensical phrases and partial sentences in English and in her native language, and she was afraid that the staff were trying to kill her. Treatment with oral olanzapine was attempted, but the patient spit out the pill and later calmed without any specific treatment. Several hours later, agitation recurred, and the patient attempted to run out of the unit. The patent was physically restrained, and treatment with intramuscular olanzapine was administered.

A diagnostic test was performed.

DIFFERENTIAL DIAGNOSIS

Dr. Judith A. Restrepo: This previously healthy young woman presented with neurologic symptoms and changes in behavior that were acute in onset and severe in nature. When behavioral changes are a prominent component of a patient's presentation, it is often helpful to cluster multiple behaviors into discrete syndromes. This patient's changes in behavior can be clustered into three syndromes — seizurelike activity, catatonia, and psychotic symptoms.

SEIZURELIKE ACTIVITY

This patient initially presented with collapse and episodes of full-body shaking, with subsequent amnesia and confusion. The blood lactate level was elevated; other laboratory test results were unremarkable, as was imaging of the head. This clinical picture is consistent with generalized seizure. During the admission to the first hospital, the patient had episodes of impaired speech, numbness of the right arm, and shaking of the right side, with an aura that was described as a feeling of dread. These features are all consistent with focal seizure localized to the left parietal lobe, predominantly the somatosensory and somatomotor cortexes.[1] Left parietal lobe seizure is associated with sensory disturbances, language impairment, and aura that may include panic attacks or an appearance of freezing, also known as "behavioral arrest."[2]

This patient's age and the semiologic features of the seizurelike events are consistent with idiopathic juvenile myoclonic epilepsy.[3] However, the progression of symptoms, despite treatment with antiepileptic medications, and the persistence of stereotyped paroxysms without epileptiform correlation on EEG are not consistent with a diagnosis of epilepsy.

CATATONIA

On admission to this hospital, the patient began to have more bizarre and less consistent behaviors, such as variable and delayed speech with occasional mutism, as well as inconsistent oppositional behaviors and repetitive, purposeless movements. Routine laboratory test results continued to be unremarkable aside from elevated levels of creatine kinase. Such findings could be compatible with the syndrome of catatonia; however, it would be helpful to know whether the patient showed other motor signs such as catalepsy or posturing. Although catatonia could explain some of her behavior changes, it can also be a feature of other syndromes, so I will focus on the third cluster of behaviors observed in this patient — psychotic symptoms.

PSYCHOSIS

The disorganized thought process and the sense of fragmentation reported by the patient are consistent with psychosis. Patients with primary psychiatric disorders that cause psychosis usually have prodromal symptoms for a year or more before the onset of psychosis. This patient had a history of mild depressive symptoms but no known history of academic or vocational decline, social withdrawal, self-neglect, or other more prominent mood or cognitive symptoms that would be typical for a primary psychiatric disorder.[4,5] In addition, new neurologic symptoms, aside from nondescript cognitive symptoms, are rare in patients presenting with a first episode of psychosis due to a primary psychiatric disorder.[4,5] Overall, a primary psychiatric disorder is unlikely in this patient.

Many medical conditions can lead to psychotic symptoms — especially when the only symptom of psychosis is disorganization. In considering the differential diagnosis for this patient's

presentation, I will use a diagnostic schema that places medical disorders into categories on the basis of disease prevalence (common or uncommon) and whether psychosis as a presenting symptom is typical or atypical, with the understanding that the most likely diagnosis will also need to account for seizure and possible catatonia.[6]

Common Disorders in Which Psychosis Is Typical

Toxidromes that result from substance use are common, and psychosis is a typical feature. Substance use may include recreational use of dextromethorphan or synthetic cannabinoids, which may not be detected on routine toxicology screening. However, substance-induced psychosis is an unlikely diagnosis in this patient given the persistence of her symptoms over the course of 10 days in an observed hospital setting.

Common Disorders in Which Psychosis Is Atypical

Cancer, stroke, and severe traumatic brain injury are all unlikely causes of this patient's presentation, given the normal imaging studies. Most endocrine and metabolic considerations are ruled out on the basis of normal laboratory test results. Although the blood level of thyrotropin was not provided for this patient, she had no other signs or symptoms of thyroid disease, which makes severe thyrotoxicosis or hypothyroidism unlikely.

The normal imaging studies of the brain make multiple sclerosis an unlikely diagnosis in this patient. No other signs of systemic lupus erythematosus, such as cytopenia, arthralgia, or rash, were observed. Similarly, no signs or symptoms suggestive of vasculitis were found.

Patients with central nervous system infections can occasionally present with psychosis. This patient's time course would be most consistent with a viral infection, such as viral encephalitis due to herpes simplex virus (HSV) or varicella–zoster virus.[7] Neurocysticercosis, a parasitic infection, is a consideration in this patient, who was born and raised in South Asia, but no characteristic cystic lesions were identified on neuroimaging.

In New England, tickborne infections such as Lyme disease, which can cause meningoencephalitis, are an important consideration. Tuberculosis is endemic in many countries in South Asia, and despite the absence of other signs or symptoms of systemic infection, performing a lumbar puncture for analysis of cerebrospinal fluid (CSF) would be useful. Human immunodeficiency virus (HIV) infection and syphilis should be ruled out, although no sexual history was provided and they are unlikely to be the cause of this patient's presentation.

Rare Disorders in Which Psychosis Is Atypical

Genetic syndromes and neurodegenerative diseases rarely manifest in the second decade of life. Wilson's disease is unlikely in this patient without signs of liver disease, which are usually present by the time of onset of psychosis. There was no reported use of nutraceuticals or traditional medicines to suggest exposure to heavy metals.

Rare Disorders in Which Psychosis Is Typical

Acute intermittent porphyria occurs in young women and is characterized not only by psychosis but also by seizure and changes in behavior. However, this patient had persistent symptoms over the course of weeks as opposed to the discrete episodes that usually characterize acute intermittent porphyria. In addition, she had no abdominal pain or other gastrointestinal symptoms. Creutzfeldt–Jakob disease is unlikely in the context of normal EEG and neuroimaging studies.

Autoimmune encephalitis is an uncommon but increasingly recognized disorder, and more than half of patients initially present with psychiatric symptoms, the most common of which is psychosis.[8,9] Associated motor changes, seizure, and catatonia commonly occur, particularly in patients with autoimmune encephalitis caused by antibodies against *N*-methyl-D-aspartate (NMDA) receptors.[8,9] Normal findings on MRI of the head can occur in up to 50% of these patients.[10] New onset of neurologic symptoms, such as the seizures in this patient, are less common than psychiatric symptoms in the initial presentation of autoimmune encephalitis. However, the co-occurrence of psychosis and possible catatonia is highly consistent with encephalitis associated with anti–NMDA receptor antibodies. To establish this diagnosis, I would obtain a sample of CSF for analysis to rule out infections that can mimic autoimmune encephalitis, and I would test both serum and CSF samples for anti–NMDA receptor antibodies,

since the presence of antibodies may be limited to the CSF in approximately 15% of patients. Because results of antibody testing are usually not available for 7 to 10 days, I would consider empirical treatment with immunosuppressive therapy.

DR. JUDITH A. RESTREPO'S DIAGNOSIS

Anti–NMDA receptor encephalitis.

INITIAL MANAGEMENT

Dr. Leah W. Morelli: Lumbar puncture was performed on hospital day 2. Gram's staining of the CSF revealed rare white cells and no bacteria. Analysis of the CSF showed 19 white cells per microliter (reference range, 0 to 5), of which 89% were lymphocytes, 8% were monocytes, and 3% were neutrophils. The CSF glucose and total protein levels were normal. Empirical treatment with intravenous acyclovir for a possible diagnosis of HSV infection was started. The blood level of thyrotropin was normal, and screening tests for HIV and syphilis were negative. On hospital day 3, results of nucleic acid testing became available; HSV DNA was not detected in the CSF, and treatment with acyclovir was stopped. On the basis of this patient's presentation and available data, a presumptive diagnosis of anti–NMDA receptor encephalitis was considered while awaiting the results of anti–NMDA receptor antibody testing.

Dr. Nagagopal Venna: In patients with presumed anti–NMDA receptor encephalitis, early treatment with immunosuppressive therapy aims to reduce circulating levels of antibodies that disrupt complex neuronal functions through interaction with glutamate NMDA receptors, which are widely distributed on cerebral neuronal membrane surfaces. Synergistic immunosuppressive regimens, including intravenous glucocorticoids and intravenous immune globulin (IVIG), are the mainstay of treatment based on clinical experience and expert opinion.[11] Treatment is started as soon as possible to mitigate progressive neurologic deterioration, which may include catatonia, dystonia, dysautonomia, central hypoventilation, and coma.[12] Treatment is usually initiated once important mimics, such as HSV encephalitis, have been ruled out. Approximately 50% of young women presenting with anti–NMDA receptor encephalitis will have an ovarian teratoma as the occult trigger of paraneoplastic disease.[13] Pelvic imaging may provide additional early support for the diagnosis while awaiting results of antibody testing. If a teratoma is detected, prompt resection is critical.

Dr. Morelli: Treatment with intravenous methylprednisolone was started for a presumptive diagnosis of anti–NMDA receptor encephalitis on hospital day 3. Abdominal imaging was performed.

Dr. Amirkasra Mojtahed: A transabdominal ultrasound image of the pelvis obtained on hospital day 5 revealed a heterogeneous mass, measuring 17 cm in diameter with mixed echogenicity, located in the right adnexa (Fig. 1A). The next day, CT of the abdomen and pelvis, performed after the administration of intravenous contrast material, showed a 17-cm heterogeneous mass in the midline of the pelvis containing fat, calcifications, and fluid and soft-tissue attenuation (Fig. 1B and 1C). The findings were consistent with an immature cystic ovarian teratoma of uncertain laterality. Additional bilateral adnexal masses were seen more inferiorly in the pelvis, which contained fat and soft-tissue elements and were more homogeneous than the larger mass (Fig. 1D). The findings were consistent with mature cystic ovarian teratomas.

Dr. Morelli: The patient was treated with a 5-day course of intravenous methylprednisolone, followed by a tapering course of oral prednisone. Treatment with IVIG was started on hospital day 6. On hospital day 7, the patient underwent left salpingo-oophorectomy and right ovary-sparing cystectomy.

PATHOLOGICAL DISCUSSION

Dr. Gulisa Turashvili: On gross examination, the left ovary was enlarged, measuring 16.5 cm in the greatest dimension, with a tan-white and diffusely nodular external surface (Fig. 2A) and a tan-white to brown-red to gelatinous yellow cut surface with hair and bone. Heterogeneous nodules and multiloculated cysts containing serous fluid were also seen. The specimen obtained from the right ovarian cystectomy measured 5 cm in the greatest dimension and revealed nodularity with a yellow cut surface and hair. The right

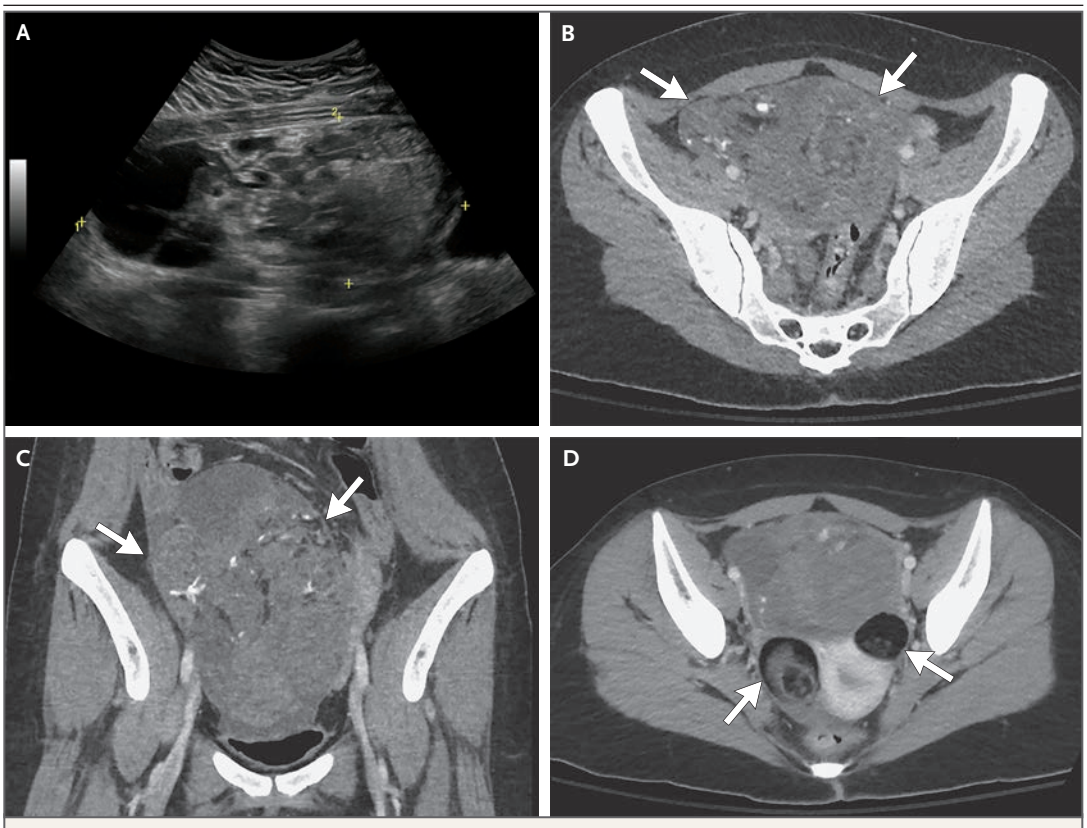

Figure 1. Imaging Studies of the Abdomen and Pelvis.

A transabdominal ultrasound image of the pelvis (Panel A) shows a 17-cm heterogeneous mass with mixed echogenicity in the right adnexa. Axial (Panel B) and coronal (Panel C) CT images of the abdomen and pelvis show a 17-cm heterogeneous mass in the midline of the pelvis containing fat, calcifications, and fluid and soft-tissue attenuation (arrows). An axial image of the pelvis (Panel D) taken at a more inferior level shows bilateral adnexal masses that contain fat and soft-tissue elements (arrows) and are more homogeneous than the larger mass.

ovarian nodules were white and firm and measured up to 1.4 cm in diameter.

Microscopic examination of specimens obtained from the left ovary, right ovarian cyst, and right ovarian nodules revealed mature tissue derived from all three germ layers (Fig. 2B). The left ovary specimen also showed diffuse sheets, primitive tubules, and pseudorosettes and rosettes of immature neuroepithelium comprising primitive-appearing cells with scant cytoplasm, hyperchromatic nuclei, and brisk mitotic activity. These features are consistent with an immature teratoma, which represented approximately 70% of the overall tumor volume (Fig. 2C and 2D). In addition, there were anastomosing channels and variably sized cysts lined by primitive cells with clear-to-eosinophilic cytoplasm, features that are consistent with a yolk-sac tumor and represented approximately 30% of the overall tumor volume (Fig. 2E). Immunohistochemical staining was positive for glypican-3 in both the immature teratoma and yolk-sac tumor components (not shown).

HOSPITAL COURSE

Dr. Jack: On hospital day 11, results of anti–NMDA receptor testing were reported. Antibodies targeting the glutamate NR1 subunit of NMDA were detected in the serum. CSF testing for NMDA receptor antibodies was not possible because of the limited amount of CSF available.

PATHOLOGICAL DIAGNOSIS

Malignant mixed germ-cell tumor.

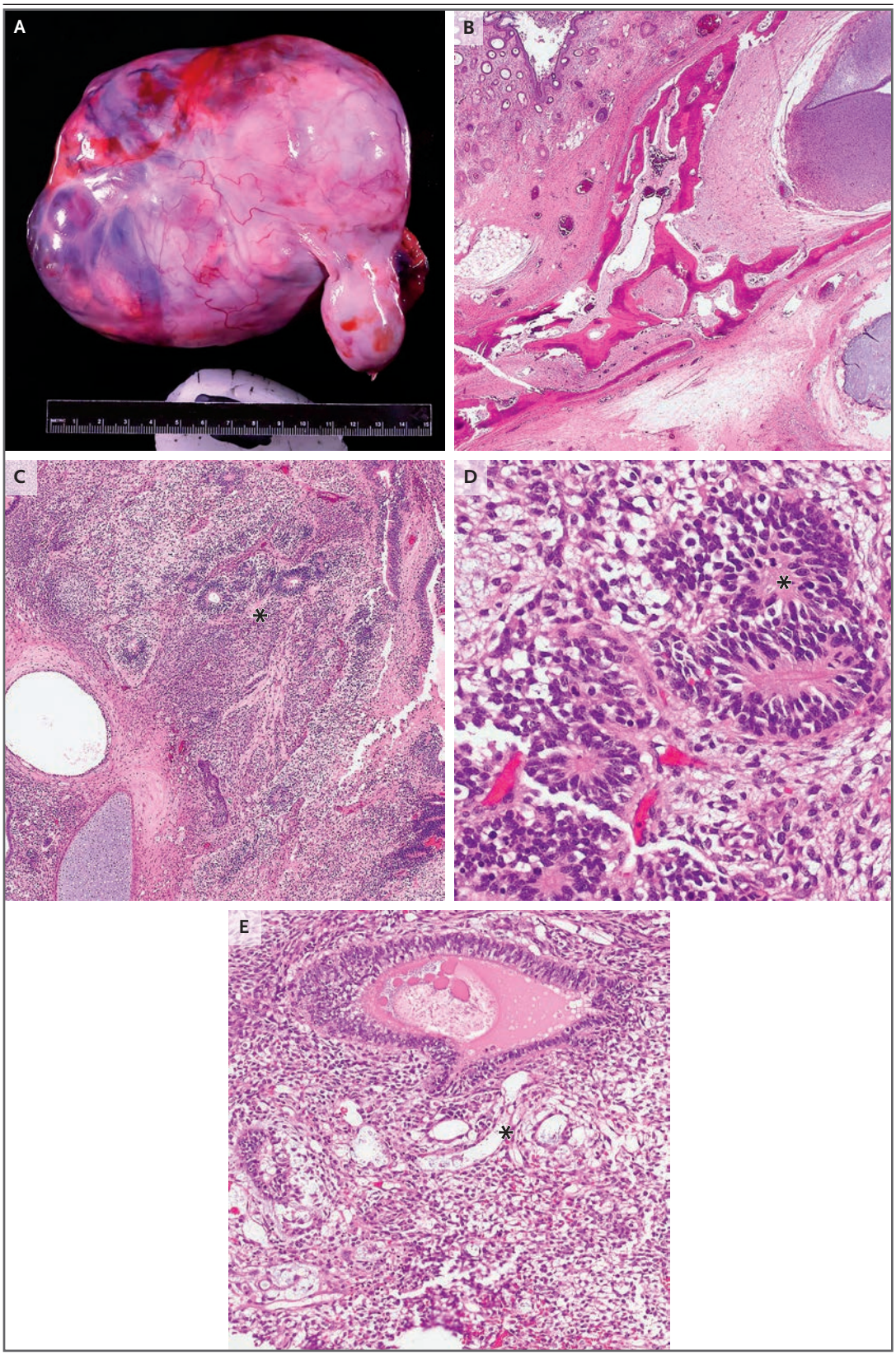

Figure 2 (facing page). Left Salpingo-Oophorectomy Specimen.

A photograph of the left ovarian mass shows a tan-white and diffusely nodular external surface (Panel A). Hematoxylin and eosin staining of a section of a mature teratoma specimen obtained from the left ovary (Panel B) shows tissue composition derived from all three germ layers, including the ectoderm (keratinizing squamous epithelium and skin adnexal structures), mesoderm (bone and adipose tissue), and endoderm (intestinal epithelium). Hematoxylin and eosin staining of a specimen of the immature teratoma from the left ovary (Panel C) shows tissue composed of diffuse sheets, primitive tubules, and pseudorosettes and rosettes of immature neuroepithelium (Panel C, asterisk); high-grade cytologic atypia with brisk mitotic activity within immature neuroepithelium (Panel D, asterisk) was present, as was a yolk-sac tumor component comprising anastomosing channels and variably sized cysts lined by primitive-appearing cells (Panel E, asterisk). The immature teratoma was approximately 70% of the overall tumor volume, and the yolk-sac tumor was approximately 30% of the overall tumor volume.

LABORATORY DIAGNOSIS

Anti–NMDA receptor encephalitis.

ADDITIONAL MANAGEMENT

Dr. Morelli: After surgery, the patient's hospital course was complicated by infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and aspiration pneumonia. Delirium developed in the context of many precipitating and perpetuating factors, and despite nonpharmacologic interventions, the patient continued to have disorientation, confusion, paranoia, agitation, and physical aggression. First-line treatment with benzodiazepines and α_2-adrenergic agonists is recommended for agitation in patients with anti–NMDA receptor encephalitis. Treatment with antipsychotic medications can lead to a paradoxical worsening of agitation, extrapyramidal symptoms, neuroleptic malignant syndrome, and catatonia, with a risk of progression to malignant catatonia.[14] When antipsychotic medications are indicated, agents with low potency are used cautiously at low doses and in combination with benzodiazepines. This patient was treated with clonidine and lorazepam, and a low dose of quetiapine was administered as needed for refractory agitation.

On hospital day 16, a clinical diagnosis of catatonia was made on the basis of observation and physical examination, aided by the use of the Bush–Francis Catatonia Rating Scale, a 23-item clinician-administered scale that helps to identify and measure the severity of catatonia.[15] It is most likely that catatonia in this patient was a manifestation of anti–NMDA receptor encephalitis, given that it occurs in up to 60% of persons with this disease.[16] However, because it developed after treatment with quetiapine, an adverse reaction to this medication may have contributed. Treatment with quetiapine was stopped, and all antipsychotic medications were avoided. Treatment of catatonia in this patient included administration of lorazepam and treatment of the underlying disease process. Patients with catatonia who do not have a response to treatment with benzodiazepines and immunotherapy may ultimately undergo electroconvulsive therapy, which has been shown to be safe and effective.[17]

Dr. Venna: This patient received additional immunosuppression in the context of worsening symptoms 2 weeks after first-line therapy.[18] Therapeutic plasma exchange was initiated in addition to induction doses of rituximab, a combination that is often used in patients who do not have a response to first-line therapy.[11]

Dr. Morelli: After the patient received additional immunosuppressive therapy and therapeutic plasma exchange, her condition improved substantially, and she showed remarkable insight into her illness without any further evidence of psychosis. After a 6-week inpatient hospitalization, she was transferred to a rehabilitation facility for ongoing speech and language therapy, occupational therapy, physical therapy, and monitoring of catatonia while lorazepam was tapered.

During the first 24 months of treatment for anti–NMDA receptor encephalitis, the risk of relapse is 12%.[9] Relapses are most likely to manifest with isolated psychiatric symptoms and can occur up to 13 years after initial diagnosis.[19,20]

Dr. Venna: In patients with anti–NMDA receptor encephalitis, immunosuppression is maintained for many months. One regimen with a favorable safety profile is administration of intravenous methylprednisolone, initially with weekly infusions, followed by infusions at 2-week inter-

vals and then at 4-week intervals over the course of 6 to 12 weeks. At her last follow-up with the neurology consultant 7 months after discharge, the patient had completed treatment with methylprednisolone. She continues to receive rituximab infusions every 6 months, and there have been no signs of relapse.

FINAL DIAGNOSIS

Malignant mixed germ-cell tumor and anti–NMDA receptor encephalitis.

This case was presented at Psychiatry Grand Rounds.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

We thank Dr. Kathy M. Tran for her contributions to the organization of this conference.

AUTHOR INFORMATION

[1]Department of Psychiatry, Massachusetts General Hospital, Boston; [2]Department of Psychiatry, Harvard Medical School, Boston; [3]Department of Radiology, Massachusetts General Hospital, Boston; [4]Department of Radiology, Harvard Medical School, Boston; [5]Department of Neurology, Massachusetts General Hospital, Boston; [6]Department of Neurology, Harvard Medical School, Boston; [7]Department of Pathology, Massachusetts General Hospital, Boston; [8]Department of Pathology, Harvard Medical School, Boston.

REFERENCES

1. Baugh L. Homunculus. Britannica (https://www.britannica.com/science/homunculus-biology).

2. Chowdhury FA, Silva R, Whatley B, Walker MC. Localisation in focal epilepsy: a practical guide. Pract Neurol 2021;21:481-91.

3. Hirsch E, French J, Scheffer IE, et al. ILAE definition of the idiopathic generalized epilepsy syndromes: position statement by the ILAE Task Force on Nosology and Definitions. Epilepsia 2022;63:1475-99.

4. Sheitman BB, Lee H, Strous R, Lieberman JA. The evaluation and treatment of first-episode psychosis. Schizophr Bull 1997;23:653-61.

5. Guasp M, Giné-Servén E, Maudes E, et al. Clinical, neuroimmunologic, and CSF investigations in first episode psychosis. Neurology 2021;97(1):e61-e75.

6. Freudenreich O, Schulz SC, Goff DC. Initial medical work-up of first-episode psychosis: a conceptual review. Early Interv Psychiatry 2009;3:10-8.

7. Granerod J, Ambrose HE, Davies NW, et al. Causes of encephalitis and differences in their clinical presentations in England: a multicentre, population-based prospective study. Lancet Infect Dis 2010;10:835-44.

8. Herken J, Prüss H. Red flags: clinical signs for identifying autoimmune encephalitis in psychiatric patients. Front Psychiatry 2017;8:25.

9. Titulaer MJ, McCracken L, Gabilondo I, et al. Treatment and prognostic factors for long-term outcome in patients with anti-NMDA receptor encephalitis: an observational cohort study. Lancet Neurol 2013;12:157-65.

10. Bacchi S, Franke K, Wewegama D, Needham E, Patel S, Menon D. Magnetic resonance imaging and positron emission tomography in anti-NMDA receptor encephalitis: a systematic review. J Clin Neurosci 2018;52:54-9.

11. Nguyen L, Wang C. Anti-NMDA receptor autoimmune encephalitis: diagnosis and management strategies. Int J Gen Med 2023;16:7-21.

12. Barter KM, Fuchs C, Graham TB, Pagano LM, Vater M. Anti-NMDAR encephalitis clinical practice guideline: improving time to diagnosis, treatment, and hospital length of stay. Neurol Clin Pract 2024;14(1):e200218.

13. Dalmau J, Tüzün E, Wu H-Y, et al. Paraneoplastic anti-N-methyl-D-aspartate receptor encephalitis associated with ovarian teratoma. Ann Neurol 2007;61:25-36.

14. Sarkis RA, Coffey MJ, Cooper JJ, Hassan I, Lennox B. Anti-*N*-methyl-D-aspartate receptor encephalitis: a review of psychiatric phenotypes and management considerations: a report of the American Neuropsychiatric Association Committee on Research. J Neuropsychiatry Clin Neurosci 2019;31:137-42.

15. Bush G, Fink M, Petrides G, Dowling F, Francis A. Catatonia. I. Rating scale and standardized examination. Acta Psychiatr Scand 1996;93:129-36.

16. Serra-Mestres J, Villagrasa-Blasco B, Thacker V, Jaimes-Albornoz W, Sharma P, Isetta M. Catatonia in N-methyl-D-aspartate receptor antibody encephalitis: phenomenological characteristics from a systematic review of case reports. Gen Hosp Psychiatry 2020;64:9-16.

17. Levy E, Reinoso P, Shoaib H, et al. Adolescents and young adults with anti-N-methyl-D-aspartate receptor encephalitis with excited catatonia: literature review and 2 illustrative cases. J Acad Consult Liaison Psychiatry 2023;64:177-82.

18. Nosadini M, Eyre M, Molteni E, et al. Use and safety of immunotherapeutic management of N-methyl-D-aspartate receptor antibody encephalitis: a meta-analysis. JAMA Neurol 2021;78:1333-44.

19. Kayser MS, Titulaer MJ, Gresa-Arribas N, Dalmau J. Frequency and characteristics of isolated psychiatric episodes in anti–N-methyl-D-aspartate receptor encephalitis. JAMA Neurol 2013;70:1133-9.

20. Gabilondo I, Saiz A, Galán L, et al. Analysis of relapses in anti-NMDAR encephalitis. Neurology 2011;77:996-9.