CASE RECORDS of the MASSACHUSETTS GENERAL HOSPITAL

Case 37-2024: A 41-Year-Old Man with Seizures and Agitation

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CME

PRESENTATION OF CASE

Dr. Lara Basovic: A 41-year-old man was admitted to the epilepsy monitoring unit (EMU) of this hospital because of seizures.

When the patient was 4 years of age, his mother noticed an episode of staring and unresponsiveness. At 19 years of age, he was the driver in a single-vehicle accident in which the car rolled over. The patient did not recall the events or seek medical evaluation after the accident. Approximately 15 years before this admission, the patient received a diagnosis of epilepsy when episodes of staring to the left and unresponsiveness occurred. The seizures were preceded by a "bad feeling" in the epigastric region and were followed by confusion, agitation, or somnolence. An electroencephalogram (EEG) reportedly revealed bitemporal sharp waves, and magnetic resonance imaging (MRI) of the head reportedly showed possible asymmetry in the temporal horns. The focal seizures initially occurred weekly and progressed to generalized tonic–clonic seizures. During the subsequent 15 years, the patient received treatment with various medications and adjusted doses of various antiseizure medications, and seizures occurred approximately once per month.

During the 3 months before this admission, the frequency of seizures increased to up to three times monthly, despite adherence to his prescribed medications, which included carbamazepine, levetiracetam, and topiramate. The patient was evaluated in the neurology clinic of another hospital, and the doses of carbamazepine and levetiracetam were increased.

Four weeks before this admission, the patient had had five seizures in 2 weeks. Two weeks before this admission, coworkers witnessed shaking of his arms and legs and called emergency medical services. The patient was transported to the emergency department of the other hospital. He received a prescription for diazepam, which was to be taken as needed at night for insomnia, and was discharged home.

The next day, the patient's neurologist was commuting to his office when he spotted the patient stumbling on the side of the road. The patient was behaving

erratically and did not respond to questions or commands. Law enforcement was called, and the patient tried to run away. He was restrained and taken to the emergency department of the other hospital. The behavior and confusion resolved, and the patient was referred to the EMU of this hospital for additional evaluation.

On the day of this scheduled admission, the patient described stress at work, inadequate sleep, and adherence to his prescribed antiseizure medications. On a review of systems, he noted that he had had "blurriness" of memory for a few days after each seizure; however, he had been able to function well at work. He had no fever, chills, malaise, or headache. During the admission interview, the patient said that something strange was about to happen. His vision became blurred, and both thighs moved nonrhythmically. The patient was aware of these events and noted that they were not typical symptoms of his seizures.

The patient had no known medical problems other than epilepsy. Medications included carbamazepine, topiramate, levetiracetam, and cholecalciferol, as well as diazepam as needed for sleep. He had no known drug allergies. The patient was born in a Caribbean country and had moved to coastal New England 5 years before this admission to live with his mother and aunt. He worked as a skilled tradesman. He did not drink alcohol, use illicit drugs, or smoke cigarettes. His two sisters were healthy, and his maternal grandmother had Alzheimer's disease. There was no family history of seizures or psychiatric disease. The results of a physical examination, including vital signs, mental status, mood, and affect, were normal.

Dr. Javier M. Romero: The initial MRI of the head revealed decreased volume of the left hippocampus and parahippocampal gyrus with increased signal intensity on T2-weighted fluid-attenuated inversion recovery imaging. Ex-vacuo dilatation of the left temporal horn was present, which was most likely secondary to the volume loss (Fig. 1A). Interictal positron-emission tomography of the head was performed, which showed subtle reduction of ^{18}F-fluorodeoxyglucose uptake in the left mesial temporal lobe (Fig. 1B).

Dr. Basovic: During the patient's first 3 days at this hospital, the doses of carbamazepine and levetiracetam were decreased gradually, and topiramate was continued. No electroclinical or electrographic seizures were observed (Fig. 1C). During the fourth and fifth hospital days, the dose of carbamazepine was further decreased, and levetiracetam was stopped. A total of five seizures were observed, with staring and subtle movements in both legs that occurred for up to 3 minutes. EEG was performed, which revealed that most electroclinical seizure events originated from the left temporal lobe focus (Fig. 2A); one event originated from the right temporal lobe (Fig. 2B). After the second seizure, the patient tried to remove the EEG electrodes and leave the room. He bit and kicked the clinicians who tried to stop him. Security personnel were called, and intravenous haloperidol and lorazepam were administered. Between seizures, the patient was calm and cooperative without agitation. The dose of carbamazepine was increased.

On the patient's sixth hospital day, the blood pressure increased to 160/100 mm Hg, and the pulse increased to 120 beats per minute. Sixteen hours after the fifth seizure, he pressed the seizure alarm button three times during episodes of leg weakness and "foggy" vision. He answered questions with one-word answers and held his hand to his chest, saying that his heart felt "off." He had auditory hallucinations of a keyboard playing and stated, "Please don't fire those three girls." Treatment with oral labetalol and lorazepam was started, and the dose of carbamazepine was further increased.

On the seventh hospital day, the patient dismantled a metal piece from the table in his room. He then held the door closed and refused to allow staff to enter and threatened them. When a staff member approached the patient, he struck that person with the metal piece. Security personnel were called, intravenous haloperidol was administered, and the patient was temporarily placed into four-point restraints. The dose of carbamazepine was further increased, treatment with levetiracetam was restarted, and risperidone was started.

The agitation resolved, and additional history was obtained. The patient explained his combative behavior by stating, "I thought they were trying to kill me," and also said, "That's not me." He was concerned about the staff member who was struck and asked if that person was hurt. He had had no previous episodes of anger or violence, although he frequently had a feeling

of fearfulness after seizures that made him want to be away from other people and on his own. He did not report auditory or visual hallucinations or paranoia, and he had never received a diagnosis of a psychiatric disorder.

During the subsequent 3 days, carbamazepine, levetiracetam, and topiramate were administered at the preadmission doses, treatment with lorazepam was continued, and clobazam was started. He intermittently declined to take medications, especially the medications that were started in the hospital, such as labetalol and risperidone. Treatment with oral carbamazepine was transitioned to intravenous lacosamide. The patient had additional episodes of foggy vision, anxiety, restlessness, impulsivity, and agitation. He persever-

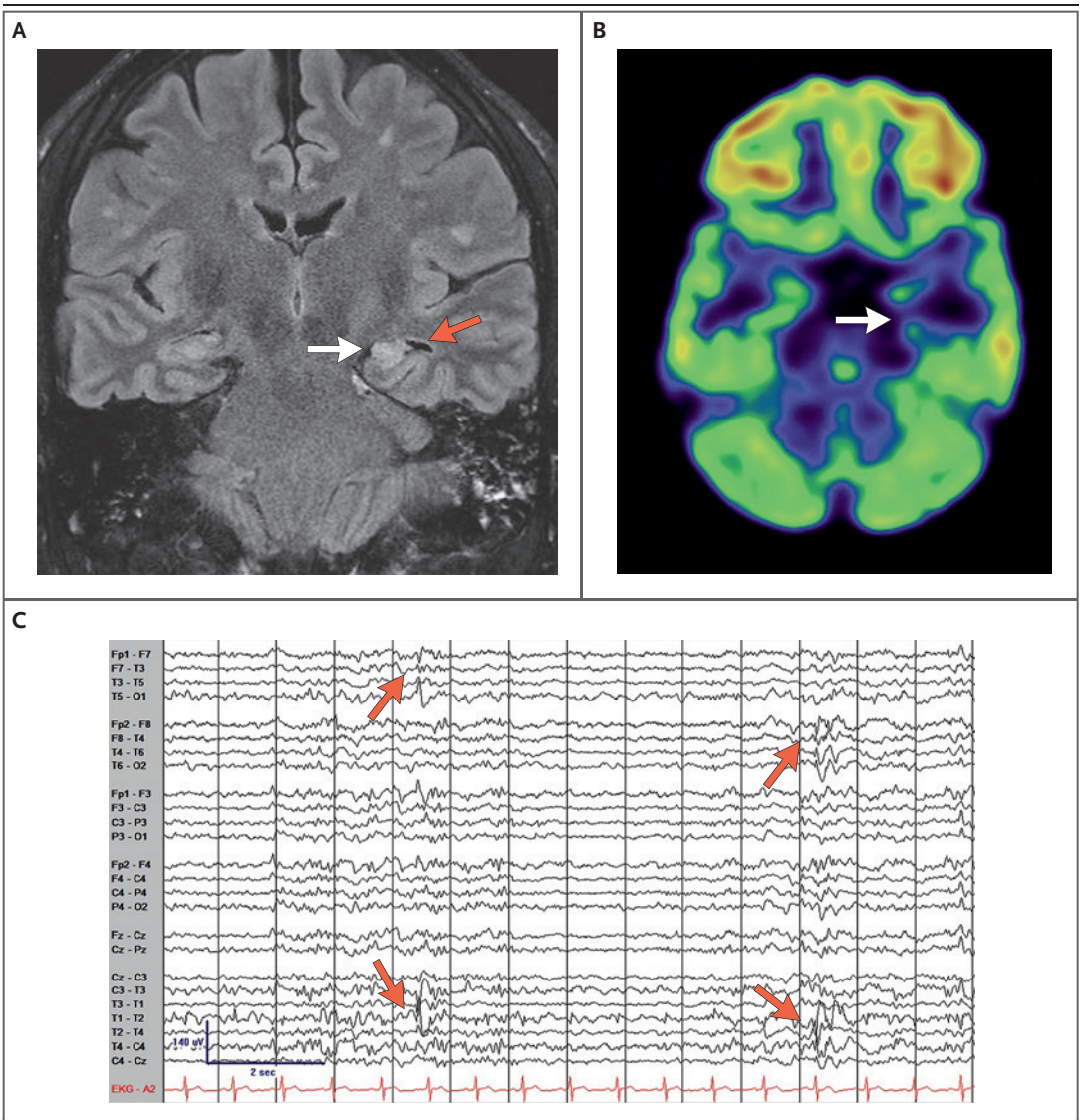

Figure 1. Initial Imaging Studies and EEG.

MRI of the head was performed on admission to this hospital. A coronal T2-weighted fluid-attenuated inversion recovery image (Panel A) reveals high signal intensity and volume loss of the left hippocampus (white arrow). Ex-vacuo dilatation of the left temporal horn is visible (orange arrow), which is most likely secondary to the volume loss. An axial image of the head obtained on ^{18}F-fluorodeoxyglucose positron-emission tomography (Panel B) shows subtle hypometabolism in the left mesial temporal lobe (arrow). The patient's baseline electroencephalogram (EEG) (Panel C), obtained on hospital day 1, shows independent left and right temporal spikes, which are visible in both the temporal chain electrodes (upper arrows) and the coronal ring electrodes (lower arrows).

ated on the idea that God had saved him. He insisted that he needed to call his mother to tell her that "it's the end." He asked her for help because he believed he would be killed in the hospital. He was seen trying to eat a mobile telephone. Two-point and four-point restraints were placed intermittently.

A diagnosis was made.

DIFFERENTIAL DIAGNOSIS

Dr. Sheldon Benjamin: This 41-year-old man with a 15-year history of apparent temporolimbic seizures, as well as a history of episodes that suggested seizures when he was 4 and 19 years of age, began to have psychotic symptoms during evaluation in the EMU. Auditory hallucinations and peculiar verbalization began 16 hours after a cluster of five seizures. Paranoid and aggressive behavior emerged in the subsequent 3 days. In addition, he had three other unexplained behaviors: aggressive behavior immediately after the second seizure while in the EMU, wandering confused on the roadside 2 weeks before this admission, and atypical seizures with preserved awareness and asymmetric nonrhythmic movements before and during this admission.

Behavioral changes in a person with epilepsy who is taking antiseizure medicines have several possible causes (Table 1). In this case, the timing of these behaviors in relation to the patient's seizures is important. This patient had both immediate and delayed postictal behavioral changes.

POSTICTAL AGITATION

Postictal delirium or agitation is more common with temporolimbic seizures than with seizures originating from other foci and tends to occur immediately after the seizure without a lucid interval. Such behaviors can be seen with or without evolution from focal to tonic–clonic seizures. Slowing on EEG and focal hypometabolism on functional MRI may be present with or without concomitant agitation. Postictal aggression is typically reactive, often occurring in response to someone touching the patient. Sometimes, the only way to confirm that the seizure has stopped and the postictal period has begun is by observing the EEG. Postictal cognitive changes have been known since ancient Babylonian times.[1] A specific form of postictal delirium that includes wandering with amnesia of the episode was called "poriomania" by Emil Kraepelin at the turn of the 20th century.[2] Since the patient was found confused and wandering on the roadside at one point before this admission, and because he attempted to leave his room in a confused, agitated state immediately after his second seizure in the EMU, the patient's behavior, at least on those occasions, was consistent with poriomania.

NONCONVULSIVE STATUS EPILEPTICUS

Status epilepticus occurs when a seizure lasts longer than 5 minutes. Focal nonconvulsive seizures with impaired awareness may manifest as confusion with repetitive behaviors that may either be subtle (e.g., blinking or twitching) or mistaken as nonseizure behavior (e.g., delirium). The presence of epileptiform or epileptic discharges on EEG and the ability to eliminate the behavior with a benzodiazepine injection distinguishes the behavior as seizures. This patient had continuous EEG monitoring in the EMU, and nonconvulsive status epilepticus was ruled out.

FUNCTIONAL OR NONEPILEPTIC SEIZURES

In addition to well-documented seizures on EEG monitoring, this patient had at least some seizures during which he was able to appropriately respond verbally while having nonrhythmic bilateral leg movements. Seizures originating in the frontal supplementary motor areas can produce bilateral leg movements, and patients might be able to respond verbally if these are focal-onset seizures. However, this constellation of behaviors could also represent functional or nonepileptic seizures, which are a type of functional neurologic disorder. A functional neurologic disorder (also known as a conversion disorder) is diagnosed when one or more symptoms of altered voluntary motor or sensory function are incompatible with a recognized neurologic condition. The *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition, text revision (DSM-5-TR)[3] does not require evidence of an adverse life event or psychosocial stressor for the diagnosis of functional neurologic disorder (functional or nonepileptic seizures). These functional or nonepileptic seizures are more common in women than men and tend to manifest in adolescence or young adulthood. Approximately 20% of people with drug-resistant epilepsy also have functional or nonepileptic seizures. Up to one third of people admitted for seizure evaluations are found to

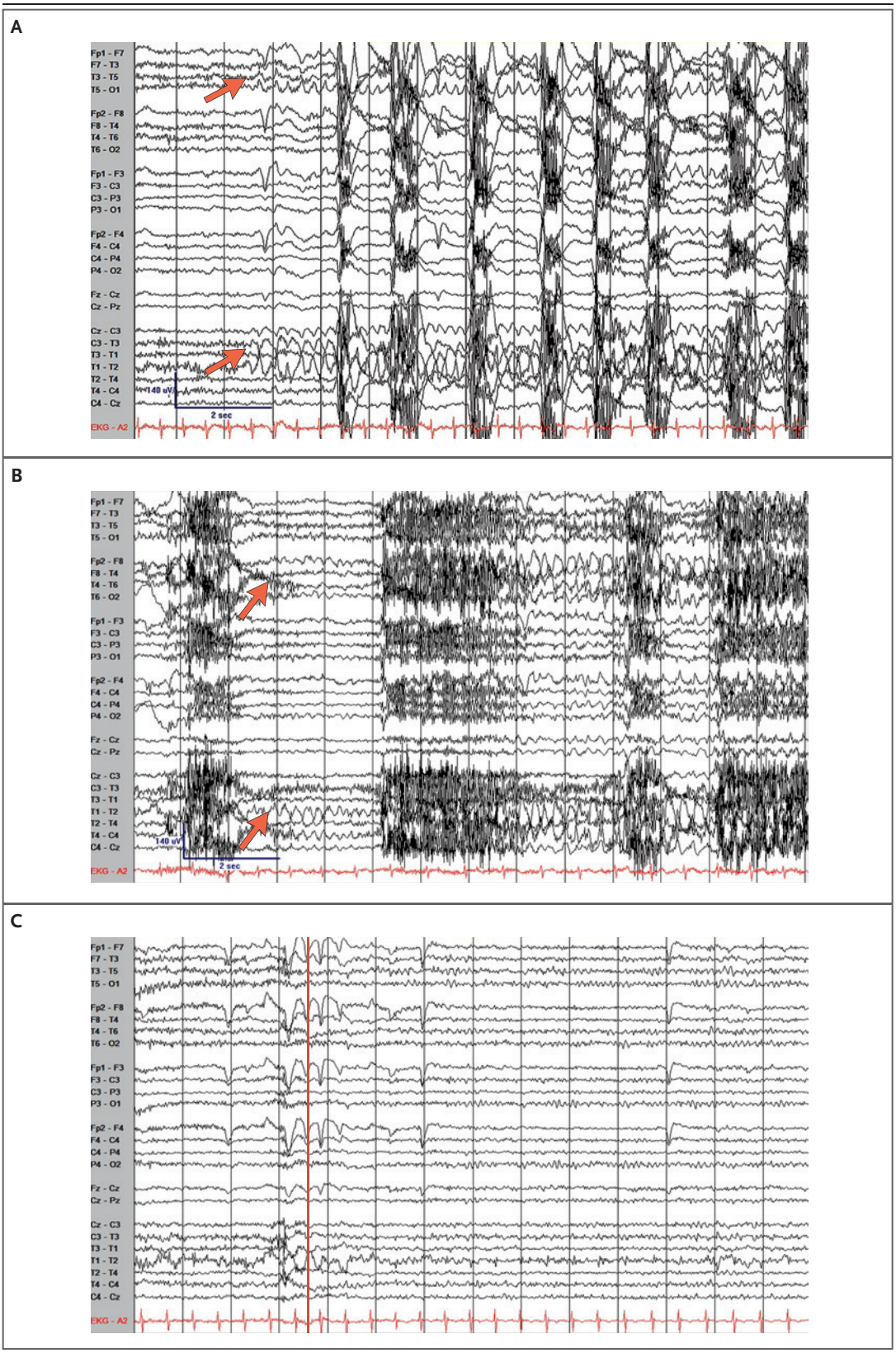

Figure 2 (facing page). EEGs Obtained during Hospital Course.

Multiple electroclinical seizures arise from the left temporal region (Panel A), and a single electroclinical seizure arises from the right temporal region (Panel B). An arrow demarcates seizure onset in each panel, in both the temporal chain electrodes (upper arrow) and the coronal ring electrodes (lower arrow). During periods of psychosis, no evidence of ictal electrographic activity is visible (Panel C). The vertical line demarcates when the patient pressed the event button to report psychotic symptoms.

have such seizures, and in one study, 7.3% of patients admitted to an EMU had both functional or nonepileptic seizures and epileptic seizures.[4,5] The diagnosis of functional or nonepileptic seizures is typically confirmed with the use of video EEG monitoring during a seizure.

PSYCHOSIS AND EPILEPSY

The risk of psychosis among people with epilepsy is 8 times that in the general population.[6] The relationship is bidirectional: the risk of epilepsy among people with chronic psychotic disorders is 2 to 3 times that in the general population. Psychotic symptoms in people with epilepsy are termed interictal if they occur without temporal relation to seizures, ictal if they are a manifestation of focal seizures, and postictal if they only occur after seizures (Fig. 3).

Interictal Psychosis

The onset of interictal psychosis tends to occur later in life than the onset of schizophrenia. Although the clinical features of interictal psychosis may sometimes appear to be similar to those of schizophrenia, negative symptoms tend to be absent, dysfunction before the onset of the disease is minimal, and affect tends to be preserved.[7]

Ictal Psychosis

Ictal psychosis is less common than interictal psychosis, but it is relatively easy to distinguish from schizophrenia. Psychotic symptoms of focal seizures last from 20 seconds to 3 minutes, tend to be stereotyped for a given person, and are not associated with persistent delusions, hallucinations, paranoia, or negative symptoms.

Postictal Psychosis

Postictal psychosis accounts for approximately 25% of psychosis diagnoses in patients with epilepsy and is more common in men than in women. Postictal psychosis tends to occur 13 to 22 years after the onset of epilepsy and is most common in patients with bilateral seizure foci who have focal impaired awareness seizures and focal seizures that become tonic–clonic seizures with bilateral seizure foci. The following diagnostic criteria[8] for postictal psychosis are commonly used: an onset of psychotic symptoms within 1 week after a return to normal mental

Table 1. Possible Causes of Acquired Behavioral Changes in Persons with Epilepsy.

Seizures (can be mistaken for nonseizure behaviors)
Behavior with a temporal relationship to seizures
Behavior associated with the lesion or gene that has also caused seizure disorder
Behavior emerging in the context of either increased or decreased seizure frequency
Subclinical epileptic activity
Cognitive–behavioral effects of antiseizure medications
Kindling*
Psychological response to chronic illness or stigma
Functional behavior change
Co-occurring or coincidental psychiatric disorder
Substance intoxication or withdrawal
Malingering

* Kindling is the theory that long-lasting behavioral changes may be caused by a mechanism similar to the observation in animal models that repetitive brain stimulation at an intensity below the seizure threshold can lead to a lowering of the seizure threshold.

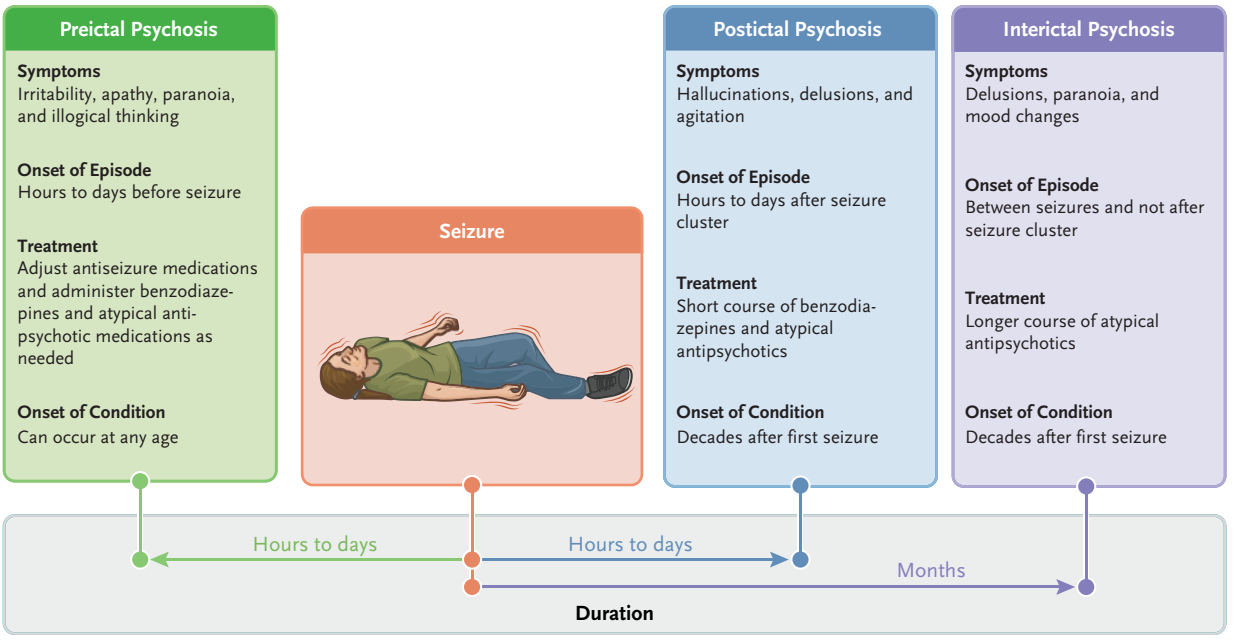

Figure 3. Timing and Characteristics of Preictal, Postictal, and Interictal Psychosis.

Shown are the symptoms, timing of onset, and treatment courses for preictal, postictal, and interictal psychosis in relation to the occurrence of a seizure.

function after a seizure or cluster of seizures; a duration of 1 day to 3 months; a clouding of consciousness or delirium, or delusions or hallucinations in clear consciousness; symptoms that are not caused by the toxic effects of antiseizure medication; no evidence on EEG of status epilepticus; no diagnosis of interictal psychosis; and no recent traumatic brain injury or substance intoxication. The onset of postictal psychosis tends to occur after a lucid interval that follows either a cluster of seizures or a seizure that is different from the person's usual seizures. Postictal psychosis, which in one study occurred in 7.8% of persons admitted to the EMU,[9] is associated with an increased risk of suicidal ideation or violent episodes. It is more likely to occur in people with interictal psychosis, and it can recur as well as evolve into interictal psychosis. Treatment targets the underlying seizure disorder.

Forced Normalization (Alternative Psychosis)

The phenomenon of forced normalization refers to an observation that in some people with epilepsy and psychosis, an epileptiform EEG will normalize when the person has psychotic symptoms and then epileptic activity will resume when the psychosis abates.[10] Convulsive therapy for psychosis was introduced in 1934, which was developed from the finding that there seemed to be an inverse relationship between epileptiform EEG discharges and psychosis. Monitoring protocols typically call for a patient's antiseizure medications to be tapered in an attempt to record seizures. Once the seizure data are collected, the antiseizure medications are sometimes reintroduced quickly and seizures are fully controlled. This rapid control of seizures may lead to concomitant psychosis associated with rapid normalization of the EEG. The forced EEG normalization that occurs with a reinitiation of antiseizure medications may explain the relatively high incidence of postictal psychosis in EMUs, and it could be a possible mechanism for the development of postictal psychosis in this patient.

SUMMARY

In this patient's case, the onset of psychosis 16 to 24 hours after a return to normal mental function following a cluster of focal impaired awareness seizures, the presence of bilateral independent

seizure foci, and the history of seizures occurring for at least 15 years and possibly 22 or 37 years before the onset of psychotic symptoms are all features consistent with a diagnosis of postictal psychosis. In addition, the patient may have had poriomania, and forced normalization of his EEG may have occurred. The DSM-5-TR diagnosis is psychotic disorder due to another medical condition (epilepsy) with delusions.[3]

DR. SHELDON BENJAMIN'S DIAGNOSIS

Psychotic disorder due to epilepsy with delusions (postictal psychosis).

HOSPITAL COURSE AND EEG STUDIES

Dr. Basovic: The patient's resting EEG continued to show bilateral temporal sharp waves and slowing, features similar to those seen on the baseline EEG recorded on hospital day 1. He had no further electrographic or electroclinical seizures. The patient-reported symptoms and behavioral disturbances had no corresponding ictal changes on EEG (Fig. 2C). However, further collateral history was obtained. The patient's family reported that he had been hospitalized for seizures approximately 5 years before the current presentation. During that hospitalization, similar psychotic behaviors had occurred for approximately 10 days.

PSYCHIATRIC ASSESSMENT AND MANAGEMENT

Dr. Caitlin Adams: According to the DSM-5-TR diagnostic criteria, this patient's symptoms are consistent with a psychotic disorder due to a general medical condition, with the specific disorder being postictal psychosis. Criteria include prominent hallucinations or delusions; evidence that the psychosis is the direct result of another medical condition; and symptoms that cannot be better explained by another mental disorder, do not occur exclusively during delirium, and cause clinically significant distress or impairment.[3] The criteria described by Logsdail and Toone in 1988 are still often used to diagnose postictal psychosis.[8]

Psychiatric disorders affect up to 50% of patients with epilepsy[11] and may include depression, anxiety, cognitive disorders, or psychosis. Risk factors for postictal psychosis include treatment-refractory epilepsy, seizure clusters (at least three seizures within 24 hours), male sex, seizures for more than 10 years, seizure aura, bilateral independent seizure foci, a history of postictal psychosis, and a family history of psychosis.[6] All these risk factors except a family history of psychosis were present in this patient. It is also important to note that the average age at the onset of postictal psychosis is older than that of primary psychotic disorders.[12] This patient had both religious delusions and fear of impending death, findings that are common in patients with postictal psychosis.[13]

Rapid recognition of postictal psychosis is crucial to prevent injury to the patient and others. Patients with postictal psychosis can be aggressive and have an increased risk of suicide. Violent behavior is more common with postictal psychosis than with interictal psychosis and occurs in clear consciousness — behavior that can be mistaken for directed violence. This patient, who had paranoia and believed that the treatment team was trying to kill him, dismantled furniture and assaulted staff.

Treatment of postictal psychosis includes the use of medications for seizures and psychosis. No prospective medication trials — symptomatic or prophylactic — have been published. Treatment decisions are based on expert consensus opinion and case reports. Verbal de-escalation is the first-line approach to resolving agitation and aggressive behavior. When verbal de-escalation is ineffective, the use of medications with a tranquilizing effect can be considered. Benzodiazepines are often used and have the added benefit of decreasing seizure activity. The use of antipsychotic medications is common, often in addition to treatment with a benzodiazepine. It is critical to distinguish between postictal psychosis, interictal psychosis, and a primary psychotic disorder, since postictal psychosis does not require long-term treatment with antipsychotics but the other conditions do.

Psychotropic medications may affect the seizure threshold, pharmacokinetics of antiseizure medications, and drug–drug interactions. The early use of antipsychotic medication may shorten the duration of the psychotic episode. Risks include possible QT interval prolongation in the short term, and if used for long-term treatment,

Table 2. EMU Protocol for Postictal Behavioral Changes.*

Before EMU Admission
Patient education on postictal behaviors
Screening for history of postictal behaviors with questionnaire
Have you ever had (or have others told you that you had) any of the following behaviors, either immediately after a seizure or in the days following a seizure or cluster of seizures?
Mood or personality changes
Increased anger or anxiety
Seeing or hearing things around you that other people could not see or hear
Feeling that others are talking about you, plotting against you, or trying to harm you
Verbally or physically aggressive behavior
If you have had any of the behaviors listed above:
What was the duration of these behaviors?
How many times have they occurred in your life?
Have you ever been treated with medications to reduce or stop these behaviors after a seizure or cluster of seizures? If so, do you recall which medication was used?
Have you ever been hospitalized because of behaviors after a seizure or cluster of seizures?
Preventive planning for patients at high risk for postictal behaviors
Start prophylactic antipsychotic medication on (or before) admission to EMU
Consider slower taper of antiseizure medication in EMU
During EMU Admission
Staged approach to initial management of postictal behaviors, which is based on symptom severity
Stage 1: Mild confusion
Calm and reassure patient
Avoid unnecessary physical contact
Increase observation by clinical team
Stage 2: Psychotic symptoms without physical aggression
Use stage 1 strategies as in stage 1
Page neurology team (and neurosurgery team, if patient has intracranial electrodes)
If patient is amenable and has no contraindications, administer atypical antipsychotic and benzodiazepine medications by mouth
Pause antiseizure medication taper
Stage 3: Physical aggression
Place four-point soft restraints and call hospital security
Administer intravenous antipsychotic and benzodiazepine medications (use intramuscular formulations if no intravenous access)
Page neurology team (and neurosurgery team, if patient has intracranial electrodes)
Consult psychiatry team
Restart home antiseizure medications

* EMU denotes epilepsy monitoring unit.

dyslipidemia and weight gain. Benzodiazepines can be used in the short-term treatment of postictal psychosis to stop seizures and decrease agitation. Considerations with respect to side effects include the risk of oversedation, respiratory suppression, and paradoxical disinhibition.

This patient received benzodiazepines and antipsychotics, and he was physically restrained to ensure his safety and the safety of those around him while his seizure medications were restarted.

PSYCHIATRIC DIAGNOSIS

Psychotic disorder due to another medical condition (postictal psychosis due to epilepsy).

DISCUSSION OF NEUROLOGY MANAGEMENT

Dr. Alice D. Lam: The EMU is a specialized inpatient unit designed for the safe evaluation of epilepsy. A common reason for EMU admission, as was the case for this patient, is evaluation for surgical management of epilepsy. In the EMU, patients with medication-refractory epilepsy undergo assessments to determine their eligibility for epilepsy surgery and the type of surgery for those who are eligible. The goal during an EMU admission is to record several of the patient's typical seizures on video EEG to determine the brain region or networks from which the seizures arise. The length of stay in the EMU is usually approximately 1 week but may be longer if seizures do not occur. To increase the probability of capturing seizures, antiseizure medications taken at home are usually tapered during the first few days of admission.

Admission to the EMU is associated with an increased risk of postictal psychosis, owing to the high proportion of patients who are admitted with medication-refractory epilepsy and the rapid tapering of antiseizure medications, which can result in seizure clusters. Although many EMUs treat patients with postictal psychosis, only a minority have a protocol for its management.[14] Our EMU did not have a protocol for the management of postictal psychosis at the time of this patient's admission; however, it was clear that we needed one. We subsequently developed a protocol, with input from nurses, EEG technologists, pharmacists, physicians (epileptologists and psychiatrists), and patients. The protocol (Table 2) outlines procedures that should be followed both before admission to the EMU (when the focus is on risk assessment, education, and prevention of postictal psychosis) and during the EMU admission (when the focus is on early identification and acute management of postictal psychosis).

Before admission to the EMU, patients are counseled on postictal behaviors and asked if they have any history of these behaviors. Those who have a history of postictal behaviors are given the option to start a prophylactic antipsychotic medication on (or before) EMU admission to reduce the risk of postictal psychosis. The inpatient EMU team is alerted about patients who have a high risk to allow for heightened vigilance and consideration of slower tapering plans for antiseizure medications.

During the EMU admission, we use a staged approach for the short-term management of postictal psychosis, which is based on symptom severity. Nurses play a key role in recognizing the early symptoms of postictal psychosis, informing physicians when they occur, and managing postictal behaviors appropriately. Training nurses to recognize the early signs, manage aggressive behaviors, and promptly administer antipsychotic and benzodiazepine medications when needed is therefore critical for the safe and effective treatment of postictal psychosis in the EMU.

FOLLOW-UP

Dr. Basovic: The patient was discharged home on hospital day 14, which was 7 days after his last seizure. In the weeks after discharge, the lorazepam, risperidone, and lacosamide doses were tapered and ultimately discontinued. Approximately 2 years later, the patient continues to take levetiracetam, carbamazepine, and topiramate. In addition, he continues to take clobazam, which was added to his regimen during his hospitalization. The patient is currently undergoing further testing before anticipated epilepsy surgery. The frequency of his seizures has decreased, with focal seizures occurring monthly and convulsions occurring only rarely. He has neither had further episodes of postictal psychosis or injury, nor had any other psychiatric symptoms.

FINAL DIAGNOSIS

Psychotic disorder due to a general medical condition (postictal psychosis).

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.

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