THE CASE

A 28-year-old woman with an extensive psychiatric history—including generalized anxiety disorder, panic disorder, and recent postpartum depression—presented with a chief complaint of right leg weakness. She stated this weakness had begun 4 days earlier. It occurred episodically and was preceded by tingling and cramping sensations. Each episode lasted a couple of minutes and spontaneously resolved. Associated with it, she experienced slurred speech and altered mentation. There was no loss of consciousness and no pain. A panic attack usually followed, consisting of feelings of impending doom, rapid breathing, palpitations, and nausea.

She had 3 prior diagnostic evaluations for this same chief complaint, twice in an emergency department (ED) and once with her primary care physician. These evaluations included lab work and extensive head imaging, which demonstrated no acute intracranial pathology. At each previous presentation, the diagnosis was an exacerbation of her anxiety disorder, and she was treated with lorazepam.

At the current presentation, her vital signs were stable. Examination revealed a notably anxious patient. She repeatedly expressed concern that she might have a brain tumor or some other deadly disease, as she had a family history of brain cancer. Her physical exam was entirely normal, including normal strength, sensation, and reflexes in all extremities.

Further head imaging (computed tomography, CT angiography, and magnetic resonance imaging of the brain) failed to reveal an etiology of her symptoms. With no clear organic cause, her medical providers again suspected an anxiety or panic episode. She was given reassurance, and an outpatient neurology consult was arranged.

THE DIAGNOSIS

One week later, at her outpatient neurology appointment, an electroencephalogram (EEG) was performed. Following photic stimulation, the EEG showed multiple right- and left-hemisphere foci of cortical hyperexcitability including a subtle sharp component (see FIGURE). Immediately following the longest of these episodes, the patient expressed a sense of anxiety and an altered sensorium similar to her prior presentations.

The EEG findings, in addition to the postictal anxiety symptoms and clinical history, were all important components that led the treating neurologist to the diagnosis of localization-related (focal) epilepsy.1 The patient was started on oxcarbazepine, a first-line anti-epileptic medication used in the treatment of focal epilepsy.2 She is being followed by a neurologist regularly and after optimizing her anti-epileptic medication, is no longer having seizures.

DISCUSSION

The difficulty of this case stems from the atypical presentation of the patient’s seizures. The key step to the correct diagnosis was a neurological consultation and an ensuing EEG.
ever, the patient received a vast spectrum of care, including multiple work-ups, prior to a conclusive diagnosis—which highlights an important issue health care providers must address.

The role of bias. From the patient’s initial visits to the ED to her hospital admission, there was a prominent affixation, known as the anchoring bias, by the clinicians providing her care: All were focused heavily on her psychiatric features. Conversely, the evaluation for patients with suspected psychiatric diagnoses should focus on successfully ruling out major organic etiology with a broad differential diagnosis. It is crucial for providers to take a step back and make a conscious attempt to avoid fixation on a particular diagnosis, especially when it is psychiatric in nature. This allows the provider to actively consider alternative explanations for a patient presentation and work through a more encompassing differential.

The distinguishing symptoms. There is a common association between comorbid mood disorders (eg, depression, anxiety) and epilepsy. Another clue is ictal anxiety or nervousness, which is commonly observed in patients with partial seizures (and occurred with our patient).

These ictal episodes can be difficult to identify within the context of an isolated psychiatric diagnosis. The distinction can be clarified by the presence of associated somatic symptoms, which in this case included unilateral cramping, paresthesia, and weakness. These symptoms should clue in a practitioner to the possibility of underlying neurologic pathology, which should prompt the ordering of either an EEG or, at minimum, a neurological consultation.

THE TAKEAWAY

This case report shows how anchoring bias can lead to a delay in diagnosis and treatment. Avoidance of this type of bias requires heightened cognitive awareness by medical providers. A more system-based approach is to have structured diagnostic assessments, such as conducting a thorough neurological exam for patients with somatic symptoms and exacerbating comorbid psychiatric conditions.

It may also help to review cases like this with colleagues from diverse disciplinary backgrounds, highlighting thought processes and sharing uncertainty. These processes may shed light on confounding diagnoses that might be playing a role in a patient’s presentation and ultimately aid in the decision-making process.

FIGURE

EEG revealed cortical hyperexcitability

The patient’s electroencephalogram demonstrated multiple right- and left-hemisphere foci of cortical hyperexcitability (boxed insets).

REFERENCES