

35 — When Epilepsy Presents as Behavioral or Psychiatric Change

Metabolic and Neurological Psychiatric Mimics · Conceptual research article · No original clinical data presented

Abstract

Epileptic activity can manifest as behavioral or experiential change rather than obvious convulsions. This creates a diagnostic problem at the border of neurology and psychiatry: a person may report fear, derealization, sensory change or confusion while the initiating event is an abnormal electrical network state. The thesis is that temporal pattern and associated neurological signs can sometimes reveal a causally distinct syndrome hidden beneath psychiatric description.

Network physiology changes the meaning of a symptom

A brief, stereotyped episode differs from continuous generalized anxiety even when both are described as 'panic.' Post-event confusion, automatisms or observed unresponsiveness can raise concern for a seizure disorder, although none is diagnostic alone. EEG and prolonged monitoring have important limitations, and a normal short recording does not automatically rule out intermittent events. The proposed causal argument must therefore integrate witness description, time course, context and testing.

An oxalate hypothesis about altered neural excitability would face a demanding comparator in epilepsy. To claim oxalate-induced seizures or behavioral episodes, investigators would have to show a credible oxalate exposure and intermediary, not simply invoke 'ionic imbalance' after an episode. Well-characterized seizure syndromes provide a stronger model of how objective network evidence can substantiate an unusual psychiatric presentation.

Time structure matters

An abrupt stereotyped episode lasting seconds or minutes differs from persistent hallucinations lasting days, though exceptions occur. Focal seizures involving temporal or related networks may produce sudden fear, déjà vu, unusual odors, visceral sensations or impaired awareness. Postictal states can include confusion, agitation or less commonly psychotic phenomena. Recurrent stereotyped events, nocturnal episodes, witnessed automatisms or new focal neurological findings make neurological evaluation more relevant.

EEG and its limitations

Electroencephalography can provide crucial evidence, but a normal routine study does not exclude epilepsy. The probability of detection depends on seizure frequency, event timing and recording duration. Brain imaging and neurological examination may be appropriate according to presentation. Conversely, an incidental EEG abnormality cannot automatically explain every psychiatric symptom, and psychogenic nonepileptic seizures require careful, non-stigmatizing differential assessment rather than dismissal.

Why medication interpretation matters

Antiseizure drugs can influence mood and cognition. Psychiatric medications can affect seizure threshold under some conditions. Drug changes, sleep loss, intoxication or withdrawal may alter the clinical picture. Treatment of an actual epileptic disorder and management of coexisting psychiatric illness can proceed together, but each requires its own diagnostic reasoning.

A bridge to inflammatory and metabolic disease

Autoimmune encephalitis, electrolyte disorders, toxic exposures and structural brain lesions may produce both seizures and psychiatric manifestations. The coexistence of two syndromes should prompt consideration of a unifying cause when the pattern warrants it, but it is also possible for them to be independent. This distinction safeguards against casually reclassifying all panic attacks as occult seizures.

Seizure phenomena can be misread as psychiatric behavior

Focal seizures may produce unusual sensations, fear, perceptual changes, automatisms or episodes of altered awareness. Some neurological events are brief and stereotyped, while psychiatric episodes often have a different temporal structure. The distinction is not absolute, and comorbid epilepsy and psychiatric illness are possible. Nevertheless, recognizing a recurrent, stereotyped spell can prevent misinterpretation of a neurological symptom as deliberate behavior or a primary psychotic experience. Detailed witness descriptions, event duration, recovery pattern and associated motor or autonomic features are often valuable.

The clinical evaluation depends on context. EEG can identify epileptiform activity, but a normal routine EEG does not exclude epilepsy. Brain imaging may identify structural contributors in selected patients, while medication history and metabolic disturbances can inform seizure risk. An acute episode of confusion after a seizure has a different causal significance from a long-standing fixed belief or a mood syndrome. When altered behavior is accompanied by loss of awareness, injuries or recurring stereotyped events, neurological evaluation is important.

Network instability as a general biological concept

Epilepsy illustrates that a change in neural network excitability can produce behaviors, experiences and emotions without any primary intention. This observation supports the legitimacy of investigating other metabolic or inflammatory influences on network regulation. However, mechanistic similarity is not equivalence. Oxalate-associated psychiatric hypotheses must demonstrate that physiologically plausible exposures actually alter neural excitability; the existence of seizures from other causes cannot fill that evidentiary gap. The most direct experiments would examine appropriate cellular electrophysiology alongside realistic concentrations and tissue transport data.

Metabolic triggers can contribute to seizures in medically vulnerable patients, including severe glucose or electrolyte abnormalities. A patient with significant renal dysfunction may have several pathways toward altered excitability. That does not imply that a person with high urinary oxalate and normal renal function has a seizure disorder. Clinical risk depends on the full neurological and metabolic setting, not a dietary category.

Distinguishing psychiatric phenomenology from episode physiology

A useful research project would classify sudden fear episodes according to their temporal pattern, associated symptoms and objective neurological findings. It could compare individuals with confirmed focal epilepsy and patients diagnosed with panic disorder, recognizing that some symptoms overlap. Careful ascertainment would reduce the risk of retrospective labeling. For patients with established epilepsy, mood and cognitive outcomes can be examined in relation to seizure frequency, medication effects and sleep, since each may influence reported psychiatric symptoms independently.

The broader terrain lesson is that neural networks are physiological systems sensitive to multiple inputs. A clinical symptom may be the visible manifestation of altered circuit dynamics rather than a simple psychological state. But the hypothesis becomes useful only when it changes the diagnostic or experimental prediction. Severe new neurological events require appropriate assessment; the presence of an interesting metabolic hypothesis is not a reason to postpone it.

The timing of a symptom is diagnostic information

A brief episode of intense fear occurring in a stereotyped pattern with altered awareness may be physiologically distinct from generalized anxiety lasting hours. A person may describe both as “panic,” but the time course, automatisms, confusion afterward and witness observations change the differential. A neurologist evaluating possible focal seizures may request EEG or imaging as appropriate, recognizing that routine tests have limitations. The goal is not to treat every panic episode as epilepsy; it is to avoid assuming that a familiar psychiatric description establishes the underlying mechanism.

Network measures can test mechanisms without proving etiologies

An EEG abnormality can support particular neurological diagnoses, but it does not explain every subjective experience. Likewise, functional brain imaging or an experimental measure of network variability cannot establish that oxalate caused a psychiatric symptom. A proposed direct neural oxalate effect would require realistic concentration ranges, adequate transport into neural compartments and reproducible electrophysiological changes. Even a positive cell experiment would not identify the clinical syndrome. A carefully sequenced research program first identifies the physiological effect and then evaluates whether it occurs in real patients.

Avoiding retrospective trigger myths

Patients often search for foods, stressors or sleep patterns preceding a seizure or unusual experience. Some triggers are clinically meaningful, but ordinary coincidence is common. If a person had a high-oxalate meal before one episode, that observation alone does not show causation. Replicated exposure–outcome patterns, controlled analyses and objective measures would be needed. The same logic applies in broader psychiatry: a plausible trigger is not a demonstrated cause until it predicts outcomes beyond chance and confounding.

Another important variable is the effect of antiseizure medications themselves. A patient's mood, fatigue or cognition may change because of seizure frequency, medication adverse effects, sleep loss or all three. If an unconventional diet changes these outcomes simultaneously, the improvement cannot automatically be assigned to a new proposed neural toxin. Longitudinal studies should record seizure control and medication changes alongside psychiatric endpoints. Otherwise a valid neurological benefit may be attributed to an unsupported mechanism, making the resulting paper less accurate even when the patient truly improved.

A future oxalate-related electrophysiology study would also need to distinguish seizure threshold from chronic psychiatric function. Demonstrating a small change in neuronal excitability in vitro does not by itself explain hallucinations, depression or autism. Researchers should move stepwise from realistic exposure to cellular response, then to validated physiological endpoints and finally to clinically meaningful phenomena. Each transition requires evidence and may fail independently.

The most important practical difference is that recurrent stereotyped spells should be evaluated as potential neurological events even when the patient uses psychiatric language to describe them. A descriptive word such as “panic” cannot settle whether consciousness, sensory processing or autonomic activity changed through a seizure mechanism. The differential depends on the complete episode and its aftermath.

Conclusion

The brain is an electrical organ, and some apparently psychiatric experiences are electrical events. The scientific gain comes from characterizing those events precisely rather than replacing all psychiatry with an unmeasured neural hypothesis.

Selected readings

- Dalmau et al., Anti-NMDA encephalitis and seizures: <https://pmc.ncbi.nlm.nih.gov/articles/PMC2607118/>
- Pollak et al., Neurological evaluation in atypical psychosis: <https://pubmed.ncbi.nlm.nih.gov/31669058/>