

36 — Delirium Is Not Primary Psychosis

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

Abstract

Delirium is an acute disturbance of attention and awareness frequently associated with medical illness, medication effects or physiological derangement. Its ability to resemble psychosis is a critical clinical fact. This paper argues that changes in timing, attention and consciousness should be interpreted as potential evidence for an active medical process before they are subsumed into an established psychiatric diagnosis.

Acute confusion calls for a causal search

A patient who becomes disoriented over hours or days after infection, surgery, medication change or organ dysfunction presents a different syndrome from a person with years of stable psychotic symptoms. Delirium can fluctuate and may feature frightening perceptual experiences, but its defining attentional disturbance changes the diagnostic pathway. Causes are often multiple: pain, sleep disruption, dehydration, metabolic abnormalities and drug toxicity can combine. No single serum marker can replace clinical assessment.

Severe oxalate nephropathy could enter the causal chain by contributing to renal dysfunction and metabolic encephalopathy. That is a biologically plausible route, especially in a documented systemic oxalate disorder. It does not follow that delirium in an otherwise well person should be attributed to dietary oxalate. The testable claim is mediated physiology: evidence of renal or metabolic disturbance temporally linked to the mental-state change.

The distinguishing features

Delirium commonly develops over hours or days and fluctuates. Inattention, disorientation, sleep-wake disturbance and changes in arousal are central features. Hyperactive delirium may look like severe agitation or psychosis; hypoactive delirium may be mistaken for depression or apathy. A person with schizophrenia may also develop delirium, so preexisting psychiatric diagnosis does not immunize against new medical illness.

Why medical causation is heterogeneous

Infection, hypoxemia, medication toxicity, withdrawal, metabolic abnormalities, organ failure, surgery, severe pain and sleep deprivation can precipitate delirium. A single neurotransmitter explanation is inadequate; systemic illness interacts with neural networks, vulnerability and environmental stressors. The correct assessment does not automatically invoke rare causes before evaluating common and dangerous ones.

Oxalate in its proper place

Severe oxalate nephropathy could contribute to renal dysfunction, and severe renal dysfunction may be associated with encephalopathy. That is a conceivable indirect pathway. The presence of delirium, however, does not justify searching for dietary oxalate as a default cause, nor does elevated urinary oxalate prove that it caused the mental-state change. Kidney chemistry, fluid status, infection and medication exposures offer more immediate and generally more probable targets for evaluation.

Consequences of the distinction

Delirium can be a medical emergency requiring prompt identification and treatment of underlying contributors and careful supportive care. Sedating an agitated patient without investigating the underlying cause may conceal clinical deterioration. Yet behavioral safety can require simultaneous symptomatic management. The right response is integrated medicine, not the artificial opposition of psychiatry and general medical care.

Attention and fluctuating awareness are diagnostic clues

Delirium is an acute disturbance in attention and awareness that often fluctuates and may involve cognition, perception, sleep and behavior. It can present with agitation, hallucinations and fear, but it also frequently presents in a quiet, withdrawn form that is easy to miss. A primary psychiatric diagnosis based solely on the most visible behavior risks overlooking infection, metabolic injury, intoxication, withdrawal, medication toxicity or organ dysfunction. The critical distinction is not whether the patient appears distressed or bizarre; it is whether there is acute alteration from baseline, impaired attention and a medically significant underlying process.

The causes are frequently multiple. An older patient with infection may also be dehydrated, sleep-deprived and taking medications that alter cognition. Treating one factor may not immediately resolve the syndrome, because the organism is experiencing several interacting burdens. This exemplifies the volume's focus on physiological context, but it should not be reframed as evidence of an undefined “toxic terrain” without diagnostic work. The responsible pathway includes history from caregivers, neurological and medical examination, relevant medication review and targeted investigations guided by the presentation.

Why the oxalate question needs this distinction

Severe oxalate-associated kidney injury can contribute to systemic illness and, if kidney function is compromised, to metabolic disturbances capable of altering mental status. A person in that state may experience delirium or encephalopathy rather than a primary psychotic disorder. This is an important plausible causal bridge and should be examined seriously. However, a patient with hallucinations and preserved kidney function cannot be assumed to have oxalate neurotoxicity simply because calcium oxalate is known to injure renal tissue. Demonstrating altered attention and systemic disease would materially change the inference.

The research endpoint should therefore distinguish delirium from psychosis, depression and anxiety. A study that pools all behavioral symptoms as “psychiatric illness” could appear to support an oxalate hypothesis because a subset of patients has kidney-mediated encephalopathy, even if the proposed mechanism has no relation to chronic psychiatric disease. This would be an analytical success only in a superficial sense; it would misidentify the biological phenotype. Precise syndromic classification is a necessary part of causal explanation.

Outcomes beyond symptom suppression

In delirium, improvement means more than a reduction in agitation. Restoration of sustained attention, orientation, sleep-wake regulation and underlying medical stability matters. Sedating medications may reduce visible disturbance without correcting the physiological cause, which illustrates why behavior is an incomplete proxy for brain function. In hospital or long-term care settings, appropriate supportive measures and the treatment of precipitating illness are central. A biochemical marker returning to normal does not alone establish that cognition has recovered.

The larger conclusion is a methodological one: serious biological psychiatry begins by asking what kind of disturbance is present. The answer determines which organ systems, time courses and medical emergencies

to prioritize. Delirium is not simply dramatic psychosis, and its recognition protects patients from both delayed medical treatment and implausible psychiatric theories.

Quiet delirium can be more easily missed than dramatic behavior

The stereotyped picture of delirium is an agitated, hallucinating patient. Yet hypoactive delirium may present as withdrawal, slowed responses and apparent depression. A patient may be described as uncooperative or uninterested when the central problem is impaired attention. Recognition depends on establishing an acute change from baseline and evaluating fluctuation over time. Family observations can be valuable, especially when a person appears superficially oriented during a brief encounter but cannot sustain attention or follow ordinary tasks. Overlooking the syndrome can delay identification of serious precipitating disease.

Why multiple medical insults matter

An older patient with an infection may also be dehydrated, have kidney dysfunction, take sedating medications and sleep poorly. None of those variables alone may fully explain the deterioration, but together they can substantially reduce cognitive reserve. This does not require imagining a mysterious toxin. It requires identifying and managing known contributors. A terrain model that refers to a “toxic load” should specify the actual clinical insults and avoid attributing delirium to an unmeasured dietary metabolite when clear medical causes are present.

The relevance of oxalate-related illness

In severe oxalate nephropathy, acute renal or metabolic complications could contribute to delirium. That is a plausible indirect mechanism with recognizable clinical intermediates. But delirium from severe renal illness must not be used as proof that ordinary high-oxalate foods cause chronic psychosis. The clinical state, magnitude of exposure and neurological endpoint differ. A useful research project would identify whether confirmed oxalate disease predicts delirium beyond the general effects of kidney injury, rather than treating any confusion in an oxalate-exposed patient as diagnostic confirmation.

Improvement in visible agitation must not be confused with restored cognition. A sedated patient may appear calmer while remaining inattentive, disoriented or physiologically unstable. Clinical outcome measures should therefore assess awareness and functional recovery as well as behavior. This principle also matters when exploring metabolic psychiatric hypotheses: a short-term change in anxiety does not establish that a presumed toxin has been cleared or that brain function has normalized. The endpoint must match the proposed mechanism, and adverse effects should remain visible in the analysis.

The importance of baseline information is often underappreciated. Family members may recognize subtle changes in attention or daily functioning long before an isolated bedside assessment can establish their significance. That information should be integrated with medical findings rather than dismissed as anecdote or treated as definitive proof of a proposed cause. A good delirium evaluation weighs history, observable cognition and physiological context together, particularly when symptoms fluctuate.

Conclusion

Delirium shows why the same outward behavior can demand radically different treatment. Its lesson for frontier medicine is to pursue mechanism with urgency and precision, not to turn every medical emergency into a favored toxin narrative.

Selected readings

- Dalmau et al., Neurological syndromes presenting with psychiatric features: <https://pmc.ncbi.nlm.nih.gov/articles/PMC2607118/>
- Bhat et al., Severe postpartum psychiatric differential: <https://pmc.ncbi.nlm.nih.gov/articles/PMC11058913/>