Guillain-Barré Syndrome in a Psychiatric Patient:

Diagnostic Pitfalls

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Guillain-Barré syndrome (GBS) is an immune-mediated polyradiculoneuropathy characterized by progressive weakness and diminished or absent stretch reflexes.[1] Early diagnosis of GBS is crucial, as early treatment decreases the duration and severity of GBS.[2] The diagnosis can be challenging, as the clinical presentation may overlap with numerous neurological or muscular illnesses. Laboratory testing usually demonstrates a typical association of elevated cerebrospinal fluid (CSF) protein level and a normal CSF cell count, known as albumin-cytological dissociation.[3] We report a case of GBS with an atypical presentation, which was initially misdiagnosed due to the patient's history of schizophrenia treated with lurasidone.

Case Report

A 59-year-old Black woman with a past psychiatric diagnosis of schizophrenia presented to the emergency department with a 2-week history of new-onset slurred speech, generalized weakness, loss of sensation, muscle rigidity, and cramping. Notably, she had been prescribed lurasidone for over 10 years, which had been effective in managing her symptoms of psychosis.

Review of the medical record revealed that the patient had presented with similar symptoms 2 weeks prior, resulting in a brief hospitalization. At that time, a stroke was ruled out, and her laboratory studies were unremarkable except for a creatinine kinase level of 459 U/L. Although her psychiatric medication had not changed in a decade and her presentation was concerning for polyradiculopathy, her symptoms were attributed to antipsychotic-induced parkinsonism based on muscle rigidity on examination. This prompted discontinuation of lurasidone and discharge to a skilled nursing facility.

During the current presentation, occurring 1 day after discharge from the previous hospital discharge, she continued experiencing significant dysarthria, ageusia, and impaired mobility. The neurological examination showed +2 motor strength in bilateral leg flexors and extensors and +1 bilateral patellar and ankle reflexes. Upon further gathering of medical history, it was discovered that the patient had received an influenza vaccine 1 month earlier.

A diagnosis of GBS was confirmed with CSF showing albuminocytologic dissociation. She received intravenous immunoglobulin with significant improvement in her symptoms.

Discussion

This case highlights how patients with comorbid psychiatric illness presenting with neurologic symptoms may experience delayed or misdiagnosis due to clinician cognitive biases. Our report further highlights the difficulties associated with diagnosing GBS.

First, individuals with mental illness face heightened risks of diagnostic errors compared to the general population due to factors such as stigma, limited access to health care, atypical disease presentations, and misinterpretation of symptoms.[4] For this patient, her history of schizophrenia and use of antipsychotic medication led to an initial misdiagnosis, delaying both diagnostic evaluation and appropriate treatment for GBS. To mitigate the risk of adverse patient outcomes, various strategies have been proposed to reduce cognitive bias and address common diagnostic errors. These strategies include educating clinicians about the prevalence of bias, allowing sufficient time for decision-making, encouraging consideration of a broad differential diagnosis before reaching a final conclusion, and promoting metacognition—the practice of reflecting on one's own thought processes.[5]

Second, diagnosing GBS poses challenges due to its diverse clinical manifestations and the need for invasive testing.[2] Our case illustrates an atypical presentation of GBS, including symptoms like loss of taste. This variability likely contributed to misinterpretation as part of her underlying psychiatric condition. In addition, antipsychotics are associated with side effects that mimic or mask the symptoms of GBS, such as muscle weakness or fatigue, complicating the diagnostic process. It is important for clinicians to distinguish between features of drug-induced parkinsonism and consider the differential diagnosis of polyradiculopathies to ensure their accurate diagnosis and treatment.

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