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Editorial

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Limbic encephalitis: complex presentation and course

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Abbreviations: VGKC, voltage-gated potassium channel; NMDA, N-methyl-D-aspartate; GAD, glutamic acid decarboxylase

Editorial

Clinicians working with children and adults will need to become aware of what appears to be increasingly more common medical and neurological disorders that present as psychiatric. One such disorder is Limbic encephalitis which is characterized by inflammation of the brain[1] caused by autoimmunity and can often present with psychosis and significant psychiatric symptoms. Although the disease is known as "limbic" encephalitis, it is seldom limited to limbic system dysfunction.[2] Limbic encephalitis is classified according to the auto-antibody that causes the disease. Many neuronal autoantibodies have been associated with Limbic Encephalitis, but the most common autoantibodies associated with it are voltage-gated potassium channel (VGKC) antibodies, N-methyl-D-aspartate (NMDA) receptor antibodies, and glutamic acid decarboxylase (GAD) antibodies.

Limbic encephalitis is often associated with NMDA antibodies. NMDA receptors are highly expressed in the forebrain, limbic system, and hypothalamus, and play a role in synaptic transmission and plasticity.[3,4] In NMDA receptor encephalitis, antibodies cause a reduction in glutamate receptors (NMDA receptors and are thought to also cause GABA-ergic dysregulation which may have the potential to contribute to or cause catatonia and psychotic symptoms in these individuals.[5] NMDA related encephalitis typically presents with psychosis, seizures, and movement disorders.[6] Initially, individuals often have more mild symptoms than PANDAS, and the initial presentation may involve seizures followed by behavior problems including inattention, temper tantrums, hyperactivity, or irritability and can develop psychotic behaviors, mutism, or unresponsiveness.[7] Though symptoms can become severe and involve inpatient hospitalization and significant time away from school, more than 75% of patients recover completely or have only mild residual symptoms.[8] Consistent with the positive prognosis is the fact that brain imaging may be normal in many cases despite significant symptoms.

Two recent cases I have assessed were initially simply more withdrawn and irritable and were seen by their primary care providers and thought to have depression and were unsuccessfully treated with SSRI's. In both cases, hallucinations eventually occurred, however, subsequent neuroleptic medication was also ineffective. One of the individuals evolved quickly into severe psychosis over a two month period and the other took 15months before psychosis and catatonia were apparent. This is generally consistent with the published literature on the topic which suggests a highly variable course of symptoms, initial diagnostic confusion, waxing and waning of symptoms, and difficulty with treatment planning, prognostications and return to productive activities.

There are many complexities to Limbic encephalitis and this includes a broad array of potential symptoms, an evolving, variable, and potentially long course of symptoms. Adding to the diagnostic complexity is the lack of definitive diagnostic testing and also the fact that disorders such as bipolar disorder and schizophrenia are being diagnosed earlier and earlier and may be considered as a differential diagnosis at some point. Managing Limbic encephalitis cases often require close communication between those providers continuously monitoring behaviors and those prescribing the medications.

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