Psychosis in Leucine-rich glioma-inactivated 1 (LGI1) antibody encephalitis: A case report

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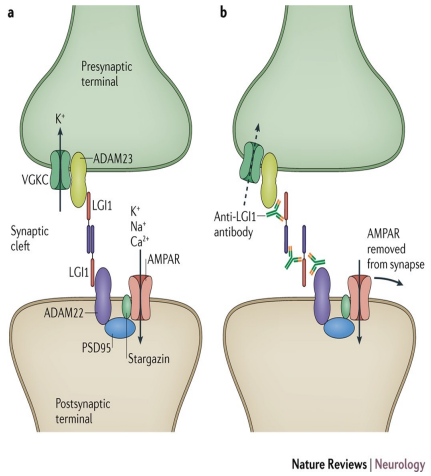
Learning objectives

1. Discuss the clinical features of Leucine-rich glioma-inactivated 1 (LGI 1) antibody encephalitis
2. Understand the mainstay of treatment for Leucine-rich glioma-inactivated 1 (LGI 1) antibody encephalitis

Background

Leucine-rich glioma-inactivated 1 (LGI1) antibody encephalitis is a rare autoimmune voltage-gated potassium channel complex (VGKC) antibody-associated limbic encephalitis. In addition to the common symptoms of limbic encephalitis such as cognitive impairment, seizures, and psychiatric disorders, this disease is also associated with faciobrachial dystonic seizures (FBDS) and refractory hyponatremia.



Brain magnetic resonance imaging (MRI) commonly but not always shows hippocampal and temporal T2 hyperintensity. LGI1 antibody encephalitis is rarely accompanied by tumors and shows a good response to immunotherapy[1]

Case

The patient is a 66-year-old male with no prior cognitive or psychiatric issues, who presented to the medical hospital initially for altered mental status and was found to have hyponatremia. Brain MRI showed increased flair signal involving bilateral medial temporal lobes. Electroencephalogram (EEG) showed generalized slowing and irregular theta waves. Cerebral spinal fluid (CSF) analysis was positive for Human Herpes Virus 6 (HHV6) antibodies and HHV6 triggered autoimmune encephalitis was suspected. The patient was treated with Ganciclovir for 3 weeks and 4 sessions of Plasma-Exchange (PLEX). He presented after a month with generalized tonic-clonic seizures, agitation, paranoia and was successfully treated with Valproic acid, Risperidone, Haloperidol, 5 sessions of Plasma Exchange (PLEX) and monthly intravenous immunoglobulin (IVIG) treatments. A few months later, he is brought in again by family with worsening aggression and psychosis. Consultation Liaison Psychiatry was consulted and on evaluation, patient was very disoriented and confabulating. CSF autoimmune encephalitis panel was noted to be positive for anti-LGI1 antibodies and a diagnosis of LGI 1 antibody encephalitis was confirmed. He was treated with intravenous immunoglobulins (IVIG) followed by Rituximab, Haloperidol and Valproic acid. Patient improved but remained with notable cognitive deficits. He was discharged to a long-term care facility.

Discussion

This case highlights that patients with LGI1 antibody encephalitis often presents with psychiatric symptoms such as cognitive deficits, behavioral changes, hallucinations, agitation, and sleep disturbance[2]. At the early stage of LGI1 antibody encephalitis, symptoms can be misdiagnosed as a psychotic disorder, major neurocognitive disorder, or even ictal psychosis. The mainstay of treatment involves steroids, intravenous immunoglobulins (IVIG), plasmapheresis and/or rituximab. Early diagnosis and prompt management is crucial in prompt recovery and improving prognosis.

Conclusion

Due to the initial psychiatric presentation of LGI1 antibody encephalitis, patients are often misdiagnosed at first and miss necessary treatment, which in turn can prolong hospital course and worsen patient outcome. As Consultation-Liaison Psychiatrists, we need to have a high index of clinical suspicion in such cases to avoid treatment delays.

References

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2. Ariño H, Armangué T, Petit-Pedrol M, Sabater L, Martinez-Hernandez E, Hara M, Lancaster E, Saiz A, Dalmau J, Graus F. Anti-LGI1-associated cognitive impairment: Presentation and long-term outcome. Neurology. 2016 Aug 23;87(8):759-65.