

**We should routinely prescribe immune modulatory therapy for patients with refractory adult-onset epilepsy who also develop psychiatric or cognitive impairment.**

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Autoimmune and inflammatory causes of epilepsy are recognized increasingly, both independently and in the context of neoplastic disease. In addition, limbic encephalitis is a complication of HHV6 infection due to bone marrow transplants. Seizures may also accompany systemic diseases such as systemic lupus erythematosus. Immune-mediated epilepsy syndromes may have a subacute onset including seizures, cognitive and psychiatric symptoms, and increased mesial temporal t2 signal. Symptoms may precede tumor detection. In non-neoplastic cases, a wide and increasing variety of antibodies have been implicated. Several mechanisms have been described. Intracellular antibodies, not directly pathogenic, such as Hu, may provoke T-cell mediated antineuronal responses. Intracellular synaptic Antigens, such as GAD65 may be targeted by both antibody and t-cell mechanisms. Cell-surface receptors, for example NMDA, may be disrupted by antibodies. The most common etiologies include anti-NMDA receptor, often associated with ovarian teratomas, anti LG1 antibodies, and anti AMPA receptors, often associated with lung, breast, or thymic tumors. In a previously healthy adult, sudden development of new onset seizures associated with acute cognitive or psychiatric symptoms is uncommon enough to create a high level of suspicion. Autoimmune encephalitis is important to recognize and treat as intervention early in its course improves prognosis. Waiting for results of serologic testing may delay appropriate therapy. Moreover, comprehensive testing should include both serum and CSF collection, which is important as well to exclude infection etiologies. Unfortunately, antiepileptic drug therapy has limited efficacy, especially for cognitive and psychiatric symptoms. Anti-inflammatory regimens have a reasonable risk-benefit profile and can be started while waiting for test results.