Perspective

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Refractory Epilepsy Associated with Anti-Ribosomal P Antibodies

Vincentiis Antonio*

Department of Neurology, Tehran University of Medical Sciences, Tehran, Iran

***Corresponding author:** Vincentiis Antonio, Department of Neurology, Tehran University of Medical Sciences, Tehran, Iran, E-mail: antoniovincentiis@gmail.com

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Description

Even though epilepsy is the third most common chronic brain disorder, little is known about the processes that cause seizures. Additionally, autoimmune diseases and epilepsy frequently co-occur, according to recent research. In the spectrum of neurological disorders characterized by the detection of neural autoantibodies in serum or spinal fluid and the responsiveness to immunotherapy, autoimmune epilepsy is becoming increasingly recognized. Seizures that are frequent or medically intractable, the presence of at least one neural antibody, inflammatory changes seen on an MRI or in serum or spinal fluid, or a personal or family history of autoimmunity all raise the possibility of an autoimmune cause. Due to electrical disturbances in the brain, epilepsy is a debilitating neurological disorder characterized by seizures sporadic electrical storms and cognitive impairment frequently without a known etiology or effective treatment. Because adjunctive immunotherapy may slow, halt, or even reverse the epileptogenic process in these patients the identification of an immune basis is crucial. Since its inception, paraneoplastic limbic encephalitis, which frequently manifests as seizures, has been associated with autoantibodies.

Chronic Brain Disorder

Outside of para-neoplastic limbic encephalitis, autoantibodies have been linked to epilepsy in other studies. Indeed, autoimmune antibodies were found in 14% of epileptics in a cohort study. In particular, the role of neural autoantibodies in chronic refractory epilepsy is being investigated. In addition, it is debatable whether the inflammatory responses in the brain or the antibodies themselves are to blame for the damage. It is essential to make a precise diagnosis of the patient's particular epileptic disorder. Antiepileptic pharmacotherapy or surgical procedures may be used to treat some forms of epilepsy; however, the type of treatment and its efficacy vary depending on the epileptic disorder in question. First-line immune therapy with corticosteroids in addition to intravenous immunoglobulin or plasma exchange should be considered when autoimmune epilepsy is suspected and clinical and serological clues suggest an autoimmune basis. If the syndrome is severe, second-line treatment with rituximab or cyclophosphamide may be considered.

The diagnosis of autoimmune epilepsy is supported by a response to immune therapy. Epilepsy is a crippling neurological condition that frequently has no known cause and no effective treatment. Antibodies or cytotoxic T cells attacking cerebral cortical autoantigens are the cause of autoimmune epilepsy, which is not uncommon and may be treatable with early-initiated immunotherapy. A favorable response to an immunotherapy and testing autoantibody profiles in serum and spinal fluid aid in diagnosis. Anti-leucine-rich glioma-inactivated 1 encephalitis can manifest as behavioral issues and focal seizures. In anti-LGI1 encephalitis, focal autonomic seizures are an alternative clinical manifestation to facial-bachial seizures. Impaired awareness as well as motor, gelastic, and focal autonomic seizures are among the other semiology of seizures that have been described. Given his age and frontal-behavioral syndrome, our patient's initial presentation strongly suggested behavioral FTD.

Limbic Encephalitis

The typical goosebumps alerted the researchers to the possibility of concurrent focal seizures and prompted them to consider different avenues of investigation. The only ictal semiology that made suspicion difficult was Girlbumps followed by a feeling of irritation. Since there was no objective sign of autonomic involvement in the neurological examination, we interpreted the recurrent goosebumps as possible autonomic auras. This might be because the episodes are so short. Rare manifestations of autoimmune epilepsy, such as focal pilomotor seizures and seizures accompanied by an autonomic aura resembling goosebumps, are frequently underdiagnosed. Due to the subtlety of the clinical findings and the negative imaging study, this case was particularly challenging. In fact, in these situations, MRI may initially be negative or nonspecific. The surface EEG was also useless. Although goosebumps may indicate temporal activity, this may be difficult to detect, and as in our case extremely brief episodes may contribute to underdiagnosis. Modern medicine has accepted autoimmunity as a fact. Immune epilepsy was recently included as one of the etiological groups in the new ILAE classification of epilepsies. A growing body of evidence indicates that drug-resistant epilepsy may have an immune-mediated pathogenesis even in the absence of limbic encephalitis or malignancy. N-methyl-D-aspartate receptor antibodies, voltage gated potassium channel

complex antibodies, collapsin response mediator protein 5 antibodies, ganglionic acetylcholine receptor antibodies, and GAD 65 antibodies are all thought to be associated with this condition. On the other hand, autoimmune epilepsy has not yet been linked to voltage-gated calcium channel antibodies.

The primary enzyme responsible for converting glutamic acid into gamma aminobutyric acid is glutamic acid decarboxylase. Anti-GAD antibodies have been linked to a number of neurological conditions, including Miller Fischer syndrome, stiff person syndrome, cerebellar ataxia, epilepsy, limbic encephalitis, and dancing eye syndrome. Anti-GAD antibodies are frequently found in conjunction with other antibodies, such as antithyroid, antinuclear, and antiparietal cell antibodies in addition to their well-known association with autoimmune disorders like type 1 diabetes mellitus. Autoimmune epilepsies are clinical syndromes in which it is thought that the immune system is responsible for the pathogenesis of seizures or for neuronal injury after seizures. The rapid onset of focal seizures, encephalopathy, cognitive decline, or other focal neurological deficits, or all of these, characterize these diseases, which typically affect otherwise healthy children. When diagnosing autoimmune epilepsies, traditional neurological diagnostics lack sensitivity and specificity, so results must be viewed in the context of a clinical setting. Because conventional antiepileptic medications are typically unable to control seizures and other neurological symptoms on their own, it is essential to take into account an autoimmune etiology as soon as possible in the clinical course to ensure the prompt initiation of immunotherapy, if necessary.