Epilepsy spectrum associated to anti-glutamic acid decarboxylase antibody

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Introduction: Anti-glutamic acid decarboxylase antibodies (GAD), initially described in type 1 diabetics, have been recently identified in some patients with epilepsy. Glutamic acid decarboxylase (GAD) antibody-associated encephalitis causes both acute seizures and chronic epilepsy with predominantly temporal lobe onset. The incidence of GAD antibody related epilepsy could be much higher than commonly believed.

Objective: The purpose of our work was to review the physiology, pathology, clinical presentation and management of GAD associated epilepsy.

Results: We included in our study 15 patients, 11 women and 4 men. The mean age of the beginning of the epilepsy was 41, 3±6 years old. All of them had pharmaco-resistant epilepsy. Neuro-cognitive disorders were found in 13 cases and movement disorders in 11 cases. A moderated lymphocytic pleocytosis was found in cerebro-spinal fluid (CSF) examination in 10 patients. Anti GAD antibodies were positive in the blood in all patients, and in CSF in 8 cases. Poorly responsive to antiepileptic drugs and moderately responsive to immune therapy with steroids, intravenous immunoglobulin and plasma exchange are obtained in all patients.

Discussion and Conclusions: Imaging and CSF evidence of inflammation along with typical clinical presentations, such as adult onset temporal lobe epilepsy (TLE) with unexplained etiology, should prompt testing anti GAD antibodies. Anti-GAD65 mediated epilepsy is often poorly responsive to antiepileptic drugs and only moderately responsive to immune therapy with steroids, intravenous immunoglobulin, or plasma exchange. Long-term treatment with more aggressive immunosuppressant such as rituximab and/or cyclophosphamide is often necessary than current immunosuppressive approaches.