Conclusions
Neuronal uptake of normal and paraneoplastic IgGs requires the interaction of the Fc portion of the IgG molecule with previously uncharacterized neuronal FcγRI receptors. Our study provides a mechanism through which antibodies reactive with intracellular neuronal proteins could gain access to their target antigens to cause neuronal injury and neurological disease. The observation that neuronal antibody uptake can be blocked by normal IgG has possible implications for patient treatment.

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Anti-Tr/DNER Paraneoplastic Cerebellar Degeneration with Marked Cerebellar and Psychological Symptoms Responsive to Plasma Exchange
Paul Crane, Don Raphael Pratt Wynn, Dana DeWitt, John Greenlee

Objective
We present a patient who developed cerebellar degeneration and severe psychological symptoms leading to the diagnosis of Hodgkin’s disease and detection of anti-Tr/DNER antibodies. The patient failed to respond methylprednisolone intravenous immunoglobulin G, rituximab, and tumor treatment but had significant improvement with plasma exchange (PLEX).

Background
Paraneoplastic cerebellar degeneration accompanying Hodgkin’s disease may have its onset prior to detection of the underlying malignancy, during its course, or following treatment. The associated autoantibody, anti-Tr, is reactive with neuronal delta/notch-like epidermal growth factor-related receptors (DNER), an autoantibody not included in all paraneoplastic testing screens. The condition characterized by progressive cerebellar injury, and response to immunosuppressive therapy and tumor treatment is generally poor.

Design/Methods
Case Presentation: A 60-year-old male presented with diplopia, progressive loss of balance, and ataxia, with impaired short-term memory, confusion, and anger outbursts. Initial commercial screen for paraneoplastic autoantibodies was negative. Two months following his initial presentation he developed inguinal lymphadenopathy. He was diagnosed as having Hodgkin’s Lymphoma Stage 1B and found by a second laboratory to have anti-TR/DNER antibodies (Titer 1:3480; Reference range <1:240), an antibody not included in the initial testing panel. CSF analysis was notable for a protein of 92 mg/dL. MRI demonstrated normal findings for age.

Results
Treatment with Doxorubicin-Bleomycin-Vinblastine-Dacarbazine (ABVD), pulse methylprednisolone, and intravenous immunoglobulin did not affect disease progression. Plasma exchange PLEX resulted in marked improvement. Symptoms worsened during subsequent treatment with intravenous immunoglobulins and rituximab but improved with further plasma exchange.

Conclusions
Although Hodgkin’s disease is an important malignancy in paraneoplastic cerebellar degeneration, its associated autoantibody is not necessarily included in commercial paraneoplastic autoantibody screens, potentially leading to delay in diagnosis. Our patient’s dramatic improvement with PLEX suggests that PLEX should be considered early in treatment, or where there is poor response to other treatment modalities.

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CaspR-2 Antibody Associated Autoimmunity in the Setting of COVID-19 (Infection, Vaccination, or Both?) and Chronic Lymphocytic Leukemia: Case Report and Review of the Literature
Neda Sattarnejad, Jamie McDonald, Anna Tomczak, Julia Sumera, Jacob Loeffler, May Han

Objective
To report a case of Anti-Contactin-Associated Protein-like2 (CaspR-2) autoimmunity in a patient with low-grade Chronic Lymphocytic Leukemia (CLL) following COVID-19 vaccination and infection.

Background
Anti-CASPR2 antibody disorder has been associated with neoplastic disorders like thymoma. Recent reports enlist COVID-19 as a potential trigger of CASPR2 autoimmunity. While the clinical presentations are similar, management differs based on the underlying etiology.

Design/Methods
We review a case of anti-CASPR2-antibody associated disorder with concurrent low grade CLL and recent history of COVID-19 vaccination and infection. Additionally, we review the literature and discuss the therapeutic challenges.

Results
A 73-years old male presented with five months of progressive fatigue, weight loss, diffuse sweating, muscle cramps, and neuropathic pain. He eventually developed bilateral upper and lower facial weakness. Patient
Anti-Tr/DNER Paraneoplastic Cerebellar Degeneration with Marked Cerebellar and Psychological Symptoms Responsive to Plasma Exchange
Paul Crane, Don Raphael Pratt Wynn, Dana DeWitt, et al.

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