A 22-year-old previously healthy Caucasian female presented with myalgia, fever, headache and cough. She tested positive for influenza A and was started on oseltamivir. Two days later, she showed mild confusion and dysarthria, followed the next day by extremities and head tremor and ataxic gait. She went to an outside hospital where cerebrospinal fluid (CSF) results were the following: red blood cells, 9,700 per mm$^3$; white blood cells 102 per mm$^3$; segmented neutrophils, 80%; lymphocyte, 14%; protein, 98.5 mg/dL; glucose 61 mg/dL. Human immunodeficiency virus and Lyme antibodies were negative. Brain magnetic resonance imaging (MRI) on admission showed cerebellar edema and hydrocephalus (Figure 1). She was treated empirically with antibiotics, acyclovir, oseltamivir, and dexamethasone 10 mg every 6 hours for 1 week. The following CSF tests were negative: bacterial and fungal cultures, venereal disease research laboratory (VDRL), Herpes polymerase chain reaction, West Nile antibodies, coccidioidomycosis antibodies, Cryptococcus antigen, and N-methyl-D-aspartate (NMDA) antibodies and cytology. Her speech remained severely dysarthric. She showed truncal ataxia, kinetic tremor in her four extremities, with dysdiadochokinesia and dysmetria. The lumbar puncture was not repeated because she developed a CSF leak. Chest, abdomen, and pelvic contrast tomography and pelvic ultrasound did not show evidence of malignancy.

Thyroid peroxidase antibodies were positive with a normal thyroid function. At day 19 from symptom onset she received 1 g of methylprednisolone daily for 5 days with minimal improvement of symptoms. After discharge, the send-out laboratory results came back for the central nervous system autoimmune panel showing elevated serum anti-glutamic acid decarboxylase (GAD) 65 titers: 0.07 nmol/L (normal <0.02 nmol/L). She was lost to follow-up for a few weeks. When she attended our clinic, her cognitive problems had completely resolved but her cerebellar symptoms remained...
similar. She was started on monthly intravenous immunoglobulin therapy (0.4 g/kg/day for 5 days, monthly); she has received it for several months with partial improvement of the ataxia. Brain MRI 6 months from disease onset showed significant cerebellar atrophy (Figure 2).

Anti-GAD-associated cerebellar ataxia affects mostly women in about 80–90% of cases, occurring mainly in their sixth decade of life. Most patients develop a chronic presentation, whereas around one-third of cases present with subacute symptoms; very rarely has it been reported as an acute condition.

Imaging studies of anti-GAD 65-related cerebellar syndrome were reported to be either normal or to indicate cerebellar atrophy. This case, with rapid onset of cerebellar symptoms and imaging evidence of active changes in the cerebellum, implies that it can present as an acute cerebellar inflammatory process than can affect young patients.
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