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Letters to the Editor

Anti-myelin oligodendrocyte glycoprotein antibody neuritis optica following anti-NMDA receptor encephalitis

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A 9-year-old girl was admitted to hospital with fever, somnolence, hemiplegia, dysphagia, and memory impairment. Cell-based assay indicated that she was positive for anti-*N*-methyl-D-aspartate receptor (anti-NMDAR) antibodies in serum and cerebrospinal fluid (CSF). On brain magnetic resonance imaging (MRI) T2-weighted (T2)/fluid-attenuated inversion recovery, abnormality was noted in the left temporal and parietal lobes (Fig. 1). Electroencephalogram demonstrated slowing of the background rhythm in the left cerebral hemisphere, but no epileptiform discharge. On lumbar puncture, 44 white blood cells had mixed cellularity and protein was 22 mg/dL. Bacterial culture and tests for various viruses were negative. Oligoclonal IgG band and anti-aquaporin-4 antibody (AQP4) were not present. The patient had no ophthalmologic abnormalities or associated tumors; she was therefore diagnosed with anti-NMDAR encephalitis and treated with three courses of pulse i.v. methylprednisolone (IVMP) and i.v. gammaglobulin (IVIG). She was symptom free at discharge and was treated with oral prednisolone (gradually tapered over 5 months). Seventeen days after cessation of prednisolone, she was readmitted due to right visual impairment, pain, and headache. The right optic disc was swollen, she had a reduced light reflex and could discern hand movement but nothing else through the right eye. Visual acuity in the left eye was intact. Brain MRI indicated contrast enhancement in the right optic nerve, but no abnormality in the cerebral hemisphere or spinal cord (Fig. 2). Serum and CSF were strongly positive for anti-myelin oligodendrocyte glycoprotein (anti-MOG) antibodies, but negative for anti-AQP4 and anti-NMDAR antibodies. There was no oligoclonal IgG band. Myelin basic protein in the CSF was elevated (149 pg/mL, standard value, <102). She was diagnosed with anti-MOG antibody-positive neuromyelitis optica spectrum disorder (NMOSD) and treated with three courses of IVMP and IVIG. Visual acuity at discharge was normal. On re-testing of stocked serum, anti-MOG antibodies were positive at the same time that symptoms of anti-NMDAR encephalitis were evident. At 18 months after initial presentation, she received oral prednisolone (5 mg/day).

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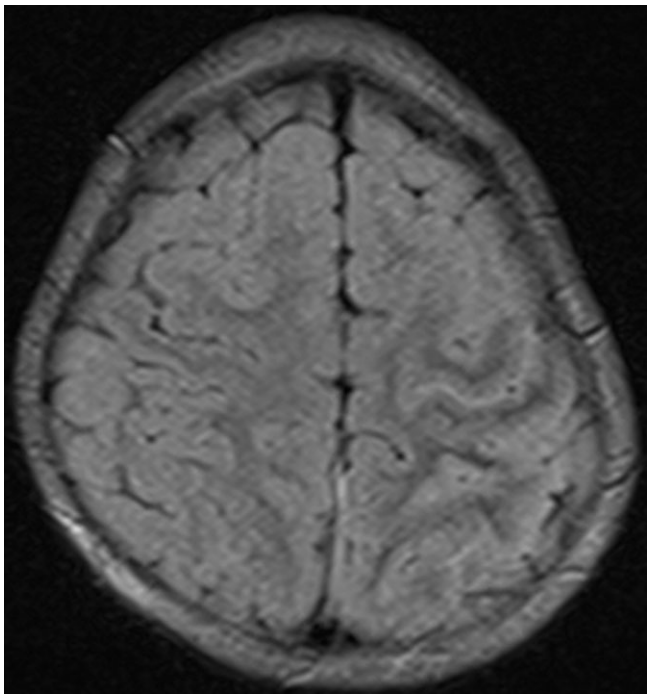

Fig. 1 T2/fluid-attenuated inversion recovery-weighted magnetic resonance imaging the brain showing high signal intensity in the left temporal and parietal lobes.

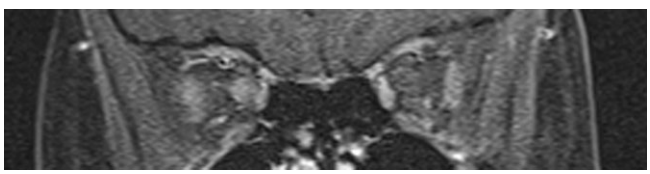

Fig. 2 T1-weighted magnetic resonance imaging of the brain showing gadolinium enhancement in the right optic nerve.

Anti-NMDAR encephalitis is an immune-mediated disorder associated with IgG antibodies against the GluR1 subunit of NMDAR.[1] It is very difficult to distinguish anti-NMDAR encephalitis from acute disseminated encephalomyelitis. Thus, further investigation of NMDAR antibody level is needed to facilitate diagnosis. Neuromyelitis optica (NMO) and NMOSD are autoimmune inflammatory disorders of the central nervous

system (CNS) characterized by acute or subacute onset of optic neuritis and myelitis.[2] Patients with NMOSD and NMO-like symptoms do not fulfill the diagnosis of NMO,[3] although they are expected to respond to the same treatment as patients with standard NMO.[4] MOG is localized to oligodendrocytes and the outer surface of the myelin sheath; thus MOG-related experimental allergic encephalomyelitis can mimic a neurological syndrome closely resembling NMO.[5] The finding that anti-NMDAR encephalitis and a demyelinating disorder may occur in the same patient is important because the treatment and outcome for each vary; therefore, physicians should check for each type of antibody. Anti-NMDAR encephalitis has a mortality rate of 7%. Anti-MOG antibody-positive NMO may be associated with severe and sustained visual loss and atrophy of the retinal nerve fiber layer.[6] Greater understanding of these types of diseases and their treatment will improve prognosis. Physicians should test for antibodies to NMDAR, MOG, and AQP4. The present case shows that immunological activation can generate anti-neuro-antibodies, resulting in various diseases. The coexistence of multiple autoimmune diseases is increasingly recognized among those with autoantibody-associated neurological syndromes,[7,8] but the mechanisms underlying autoimmunity and autoantibody production are unclear.[8] Future studies should identify factors that predispose individuals to develop autoantibodies against multiple CNS antigens.

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Disclosure

The authors declare no conflicts of interest.

Author contributions

K.Y., M.H., and A.Y. designed the study; K.Y. and M.H. collected and analyzed data; K.Y. wrote the manuscript; M.H. and A.Y. gave technical support and conceptual advice. All authors read and approved the final manuscript.

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Pediatrician role in public health: Domestic animal-related injury alerts for neonates

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My colleagues and I had previously reported on a neonate with meningitis and bacteremia caused by *Pasteurella multocida* following a cat scratch.[1] *P. multocida* is a Gram-negative coccobacillus that is part of the usual human oral flora or is found in the nails; moreover, it exists in many animals, especially cats.[2] In the previous study, a 23-day-old girl presented with decreased appetite and irritability for >2 days. Eighteen