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Deterioration of clinical features of a patient with autism spectrum disorder after anti-N-methyl-D-aspartate receptor encephalitis

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ANTI-N-METHYL-D-ASPARTATE RECEPTOR (NMDAR) encephalitis is an autoimmune disorder that presents with psychiatric symptoms, seizures, dyskinesia, and autonomic instability.[1] To our knowledge, there is no report of an anti-NMDAR encephalitis patient with a previous psychiatric history, such as autism spectrum disorder (ASD). Here we describe the clinical course of an adult Japanese woman who may have had anti-NMDAR encephalitis, in addition to ASD.

A 23-year-old woman was transferred to our hospital for the treatment of periodic loss of consciousness. She was born via vaginal delivery at 36 weeks' gestation with a birthweight of 2450 g. She did not show any delay in the development of motor and language skills. After she entered kindergarten, however, her mother noticed her social skill problems and unusual interests/behaviors. She preferred to play alone, seemed not interested in others, and was easily upset by minor changes in routines resulting in aggressiveness toward her mother. Despite these problems, her parents did not consult local health centers or pediatric clinics because she did very well academically.

At the age of 12, the patient underwent resection of an ovarian teratoma. At 14, she experienced acute-onset behavioral changes, including visual hallucinations, persecutory delusions, irritability, and agitation. She was admitted to a local psychiatric hospital. She showed no abnormal movements, such as catatonia or dyskinesia. Her psychiatric symptoms rapidly subsided with risperidone (6 mg/day) treatment. She was discharged for home 1 month after admission. However, the social skill problems and aggressiveness toward her mother remained and actually worsened after the psychosis. From the age of 14 to 23, she was treated with risperidone as an outpatient.

At 23, she began to show frequent atonic attacks with consciousness loss. Two months later, she was admitted to our hospital. Her head magnetic resonance imaging and single-photon emission computed tomography results were all normal. Repeated electroencephalography examinations showed diffuse 3- to 7-Hz waves. The atonic attacks were successfully treated with phenytoin. Cerebrospinal fluid and serological analyses, including viral DNA (herpes simplex, varicella-zoster, and measles) and autoantibodies (anti-NH_2-terminal of α-enolase and anti-NMDAR), revealed no changes in any of the markers examined except for an elevation in the titer of anti-NMDAR antibody. We diagnosed her as having anti-NMDAR encephalitis. In addition, the diagnosis of ASD was confirmed by her result on the Autism Diagnostic Interview-Revised.[2] Although immunotherapies might have been useful for the anti-NMDAR encephalitis, the patient and her parents refused immunotherapy. Her aggressiveness was improved by behavioral analysis approaches. She was discharged ~100 days after admission without severe subsequent complications.

Our experience suggests that anti-NMDAR encephalitis can worsen both the core features and peripheral symptoms of ASD.

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