Anti-ma2 receptor encephalitis mimicking Huntington chorea

Sir,

Anti-ma2 receptor encephalitis is an immune-mediated disorder of brain presenting with movement disorders such as chorea, dyskinesia, loss of consciousness, language dysfunction, seizures, psychosis, and autonomic instability. This disorder includes a wide range of neuropsychiatric manifestations which overlap with presentations of many other neurological diseases. For instance, choreiform movements and psychiatric symptoms in anti-ma2 receptor encephalitis could be similar to Huntington disease, and hence the possibility of misdiagnosis should be considered before treatment.

Herein, we present an anti-ma2 receptor encephalitis case with initial Huntington-like symptoms. A 71-year-old man referred to the emergency department of Al-Zahra Hospital (Isfahan-Iran) with loss of consciousness. His relative mentioned a history of progressive choreiform movement in the distal of upper limbs two years ago which was gradually accompanied by neurologic manifestation as amnesia. In addition, hyperlipidemia, diabetes mellitus, and hypertension were noted in the medical history of the patient. He has been taking metoprolol, metformin, aspirin and hydrochlorothiazide. In addition, sodium valproate, haloperidol, and levetiracetam were used for treating his chorea. On arrival, the patient was in stupor state, vital signs were stable, and was not febrile. Hyponatremia was noted in laboratory data which was treated; slight improvement was obtained in the level of consciousness of the patient but still he was in drowsy state. Magnetic resonance imaging (MRI) showed small vessel disease in basal ganglia and subcortical regions and brain atrophy of the temporal lobe with dilation of temporal horns of lateral ventricle accompanied by hyperintensity in both hippocampi and amygdale. Cerebrospinal fluid examination was normal except for mild elevation of protein. Polymerase chain reaction for herpes simplex virus was requested. Genetic test for Huntington chorea was negative for him and his family. With suspicious of viral encephalitis, he was treated with acyclovir but there was no improvement, and also all tests for infections were negative. Hence, autoimmune panel was requested, and serum was positive for anti-ma2 receptor antibody which led to the diagnosis of autoimmune encephalitis. Workup for paraneoplasia was done including high-resolution computed tomography, prostate-specific antigen, and testis sonography, which were all negative. Hence, the patient was treated with corticosteroid and plasma exchange. After a week, a significant recovery in his consciousness and partial improvement in chorea were noted and he was discharged with oral prednisolone. Three months after treatment, he made substantial recovery of chorea, and follow-up examinations revealed no evidence of underlying cancer. This is the first report of anti-ma2 receptor encephalitis misdiagnosed with Huntington disease. Despite the Huntington-like prodromes, loss of consciousness and brain MRI suggested autoimmune encephalitis in our patient which was confirmed by the detection of anti-ma2 receptor antibody in serum. On admission, our patient presented with hyponatremia which is observed in approximately 60% of patients with autoimmune encephalitis.1 We illustrated the importance of predicting similar prodromes of anti-ma2 receptor encephalitis and Huntington chorea in this report.

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Conflicts of interest
There are no conflicts of interest.

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