

Hyperkinetic Movement Disorder in a Girl with Anti-NMDA Receptor Encephalitis

An 8-year-old girl previously in good health presented with history of five days of acute onset confusion, impaired speech, lack of sleep, two episodes of brief generalized seizures and involuntary movements. On examination, she was completely unaware, agitated, and mute. She had peculiar repetitive, rapid, eyelid, ocular and oro-facial dyskinesias, accompanied with continuous choreo-athetoid movements (*Fig. 1 and Web Video 1*). Neuroimaging was normal. Cerebrospinal fluid was acellular with normal proteins and sugar. A diagnosis of Anti-N-methyl-D-aspartate (anti-NMDA) receptor encephalitis was confirmed by demonstration of CSF anti-NMDA receptor antibodies. She was initiated on methylprednisolone (30 mg/kg/day for 5 days) and intravenous immunoglobulins pulse (1g/kg, for 2 days). She responded remarkably to treatment with cessation of all movement abnormalities and full recovery of consciousness, speech, cognition and sleep pattern. She started attending regular school after a month of treatment.

In anti-NMDA receptor encephalitis, oro-lingual-facial dyskinesias are the most characteristic movement abnormality. This movement disorder frequently occurs along with limb and trunk choreoathetosis, oculogyric crisis, dystonia, rigidity, and opisthotonic postures. This pattern of movement disorder is important to recognize



FIG. 1 Movement disorder in anti-NMDA receptor encephalitis. (See video at website).

because this disorder is a treatable cause of severe neuro-morbidity. Resembling movements disorders can occur in children due to drugs or toxins, basal ganglia involvement in viral encephalitis, Sydenham chorea, neurometabolic disorders, or hypoxic/ischemic insult to the basal ganglia. Symptomatic treatment of the hyperkinetic movement disorder may be difficult but drugs like valproate, haloperidol, benzodiazepines, and tetrabenazine have been tried.

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