Movement disorders in anti-NMDA receptor encephalitis

Sir,

A 15-year old boy was admitted after an initial episode of generalized tonic–clonic seizure. Brain computed tomography and magnetic resonance imaging failed to reveal any lesion. He returned home with levetiracetam therapy but was readmitted 2 days later after developing dysphasia, dyspraxia, and neuropsychiatric symptoms (panic, agitation, hallucinations). The electroencephalogram ruled out epileptic disorders.

Common causes of viral or bacterial meningoencephalitis were excluded by cerebrospinal fluid analysis. The diagnosis of anti-N-methyl-D-aspartate receptor (anti-NMDAR) encephalitis was made on the basis of the detection of specific antibodies in the serum and cerebrospinal fluid. A treatment with pulse methylprednisolone was started (1 g daily over 5 days, with a progressive decrease over 2 weeks) and the treatment was combined with plasma exchange. From day 7, the patient developed a large variety of abnormal movements [Video 1]. These manifestations were partially controlled by sedative (lorazepam) and neuroleptic (risperidone) drugs and disappeared at the end of the plasma exchange sessions. The patient was further investigated by total body positron emission tomography (PET), and there was no evidence of a testicular teratoma.

Movement disorders typically appear after a period of prodromal and psychiatric manifestations in adults, however, they may be the initial manifestation in children with anti-NMDAR encephalitis.[1-3] The incidence of movement disorders may be as high as 86% in adults and 84% in children.[4] They should be clearly differentiated from epileptic manifestations. Chorea is particular frequent in younger children, along with orofacial dyskinesias.[4,5] Stereotypic movements of the limbs and trunk are also commonly described, whereas opisthotonus seems less frequent.[4,5] In a series of 32 children and adolescents with anti-NMDAR encephalitis, stereotyped movements and orofacial dyskinesias had a respective incidence of 85 and 45%.[6] All these hyperkinetic movements usually improve together with psychiatric manifestations after the introduction of immunosuppressive therapy.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

Emilie Servais[1], Sophie Fastre[2], Philippe Hantson[1,3]

[1]Department of Intensive Care Medicine, Cliniques St-Luc, [3]Louvain Centre for Toxicology and Applied Pharmacology, Université catholique de Louvain, [2]Department of Neurology, Cliniques de l'Europe, Brussels, Belgium

Address for correspondence:
Dr. Philippe Hantson,
Department of Intensive Care, Cliniques St-Luc, Avenue Hippocrate, 10, 1200 Brussels, Belgium.
E-mail: philippe.hantson@uclouvain.be

References

1. Vincent A, Bien CG. Anti-NMDA-receptor encephalitis: A cause of psychiatric, seizure, and movement disorders in young adults. Lancet Neurol 2008;7:1074-5.
2. Dalmau J, Lancaster E, Martinez-Hernandez E, Rosenfeld MR, Balice-Gordon R. Clinical experience and laboratory investigations in patients with anti-NMDAR encephalitis. Lancet Neurol 2011;10:63-74.
3. Suthar R, Saini AG, Sankhyan N, Sahu JK, Singhi P. Childhood anti-NMDA receptor encephalitis. Indian J Pediatr 2016;83:628-33.
4. Baizabal-Carvallo JF, Stocco A, Muscal E, Jankovic J. The spectrum of movement disorders with anti-NMDA receptor encephalitis. Mov Disord 2013;28:543-7.
5. van de Riet EH, van Bronswijk SC, Schieveld JN. Movement disorders in anti-N-NMDA receptor encephalitis. Tijdschr Psychiatr 2015;57:109-13.
6. Florance NR, Davis RL, Lam C, Szperka C, Zhou L, Ahmad S, *et al.* Anti-N-methyl-D-aspartate receptor (NMDAR) encephalitis in children and adolescents. Ann Neurol 2009;66:11-8.

Video Available on: www.neurologyindia.com

Access this article online	
Website: www.neurologyindia.com	Quick Response Code
DOI: 10.4103/neuroindia.NI_739_16	

How to cite this article: Servais E, Fastre S, Hantson P. Movement disorders in anti-NMDA receptor encephalitis. Neurol India 2017;65:632-3.