P148

A treatable parkinsonism: lesson learned from a case of NMDARE autoimmune encephalitis

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A 25-years-old man without any previous relevant medical history referred to our outpatient clinic for a few days progressive onset of slowness of movements and muscle rigidity. Three months before he was admitted to a psychiatry service for visual hallucinations, episodes of nonsensical speech and acute paranoia with aggressiveness complicated by a tonic-clonic seizure during the hospital stay. He received a diagnosis of psychosis and was treated with haloperidol (6 mg/die) and benzodiazepines. At the admission, neurological examination showed catatonic abnormal postures, hypomimia, generalized bradykinesia of upper and lower limbs (left more than right) and postural tremor of the left hand (video 1). Brain MRI was normal while EEG showed periodic lateralized epileptiform discharges in the right temporal region. In the contest of a new onset of psychosis, seizure, rapidly progressive parkinsonism, with normal brain MRI and EEG abnormalities a diagnosis of possible autoimmune limbic encephalitis was considered. Cerebrospinal fluid analysis revealed slight increases of protein level and IgG index, a normal cell count and antibodies against NR1/NR2 heteromers of NMDA-receptor. No systemic tumour was identified by combined CT scan with 18F-fluorodeoxyglucose PET. He was started on intravenous IgG and methylprednisolone that resulted in a complete and rapid improvement within four days of both parkinsonism and psychiatric symptoms (video 2). Early recognition of a rapidly immune-mediated parkinsonism caused, in our case, by either autoimmunity against the basal ganglia and intolerance to neuroleptics is crucial because of the potential for recovery of signs and symptoms after first line immunotherapy [1].

References:

[1] Balu R, McCracken L, Lancaster E, Graus F, Dalmau J, Titulaer MJ. A score that predicts 1-year functional status in patients with anti NMDA receptor encephalitis. Neurology. 2019.