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LETTERS TO THE EDITOR

Antipsychotic-induced akathisia and neuroleptic malignant syndrome in anti-NMDAR encephalitis

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KEYWORDS: antipsychotic, NMDAR, akathisia

TO THE EDITOR:

In a recent review in *Annals of Clinical Psychiatry*, Kruse et al[1] highlighted the importance of recognizing psychiatric symptoms as a common early feature of anti-*N*-methyl-D-aspartate receptor (NMDAR) encephalitis and the challenges in managing these symptoms while waiting for a patient to respond to immunotherapy. We present 2 cases to highlight the risk of extrapyramidal symptoms (EPS) in patients with anti-NMDAR encephalitis who were treated with antipsychotics for psychiatric symptoms.

Case 1

A 4-year-old boy presented with a 4-week history of aggressive behavior, hallucinations, and seizures. His cerebrospinal fluid (CSF) had 30 mononeuclear cells/μL and was positive for IgG antibodies against NMDAR. He was treated with 2 g/kg of IV immunoglobulin (IVIg) and 3 days of IV methylprednisolone, 30 mg/kg/d. Distressing episodes of aggressive behavior, mood swings, and hallucinations persisted despite receiving nitrazepam, 2.5 mg twice daily. Quetiapine was introduced, 5 mg in the morning, and when the drug was increased to 10 mg/d the boy was noted to have continuous restless leg and body movements that were worse during sleep over 2 nights. He had a constant urge to keep moving during the day. The phenomenology was suggestive of akathisia. Quetiapine was stopped, and the akathisia resolved within 2 days. His ongoing disease was treated with 3 doses of rituximab (375 mg/m^2) and further IVIg 2 g/kg with complete recovery over the ensuing 12 months.

Case 2

A 13-year-old male presented with lethargy, visual hallucinations, behavioral change, dystonia, stereotypical movements, and increasing agitation. CSF showed pleocytosis (13 cells/μL) and autoimmune encephalitis was suspected and he was treated with IV methylprednisolone, 30 mg/kg/d for 3 days. Retrospective testing of acute serum and CSF was positive for NMDAR antibodies. Worsening visual hallucinations, agitation, dystonia, and stereotypical writhing movements were treated with droperidol, 2.5 mg twice daily (0.05 mg/kg/dose) and benzotropine, 1 mg twice daily (0.02 mg/kg/dose). After 3 days on droperidol, he developed hypertension, sweating, increased rigidity, and elevated creatine kinase. Neuroleptic malignant syndrome (NMS) was suspected, droperidol was stopped, and benzotropine continued. The patient's NMS resolved within a few days of stopping droperidol. After a 3-month admission, the patient made a complete recovery and has been well for 5 years.

DISCUSSION

Antipsychotics and other psychotropics often are administered in cases of anti-NMDAR encephalitis with associated psychiatric symptoms with poor efficacy[1] and can precipitate or exacerbate motor symptoms.[2,3] It can be challenging to differentiate EPS of antipsychotics from the movement disorders seen in anti-NMDAR encephalitis, as highlighted by previous descriptions of NMS-like syndrome in anti-NMDAR encephalitis,[2,4] even in the absence of antipsychotic administration. In the 2 cases we describe, onset of new motor symptoms with antipsychotic initiation, worsening of motor symptoms with increased doses of antipsychotics,[2] and improvement in symptoms when the antipsychotic was stopped supports the hypothesis that these symptoms were medication effects rather than part of disease evolution alone. In both of our cases, akathisia and NMS were clearly associated with starting antipsychotics, and resolved on stopping the respective medication. Interestingly, anecdotal reports have described higher prevalence of adverse reactions to antipsychotics in Sydenham chorea, another autoimmune movement disorder.[5] These observations

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suggest a vulnerability to classic neuroleptic side effects in patients with anti-NMDAR encephalitis. ■

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