Risperidone-Induced Tardive Dyskinesia in an Autistic Child

To the Editor: There are over a dozen cases of reported risperidone-induced tardive dyskinesia in the literature, with the vast majority occurring within 1 year after starting the neuroleptic.[1] However, in several of the case reports there was no cause-and-effect relationship of risperidone and tardive dyskinesia.[2] The objective in presenting this particular case is to highlight the challenges in detecting risperidone-induced tardive dyskinesia in the medical setting.

Case report. A 13-year-old boy with a history of autism, obsessive-compulsive disorder (OCD), and attention-deficit/hyperactivity disorder (ADHD) was evaluated over a 6-month period for abnormal gait and truncal movements. The patient was prescribed risperidone 4 years earlier to address behavioral issues. He was initially started on risperidone 0.25 mL twice a day; the dose was increased over subsequent years to 1.75 mL twice daily. However, 6 weeks after the last dose increase, the patient began to exhibit signs of an abnormal gait, insomnia, and aggression. Moreover, in the summer of 2017, his parents noticed an occasional foot slap gait, which became more frequent over time.

Three months after onset of the foot slap gait, the patient developed a slight limp. In the fall of 2017, the patient began ambulating with excessive inversion of the right foot and arching of the trunk. He was evaluated by a school physical therapist who felt the symptoms were related to sensory issues. The therapist recommended a weighted vest to help with proprioception. A month later, the patient developed involuntary truncal movements and was unable to maintain the standing position without continuous hand support. In addition, there was noticeable facial grimacing but no blepharospasm. He was seen several times by his pediatrician who ordered x-rays including alignment films to assess for possible leg-length discrepancy. No rating scales were used to assess the severity of the dyskinetic movements.

The imaging studies revealed a mild pelvic obliquity with mild compensatory dextroscoliosis. The patient's physical symptoms and aggression continued to get worse, and the pediatrician prescribed benztropine to help ameliorate his dyskinetic movements. However, benztropine had little effect in resolving or reducing the patient's physical symptoms and actually increased his OCD behavior. After a short trial of benztropine, the pediatrician recommended a slow-taper protocol for risperidone. In addition, the patient was prescribed clonidine 0.1 mg and fluoxetine 10 mg to address his increased aggression and behavioral issues. Risperidone was discontinued over a 4-week period. A neurology consult confirmed iatrogenic tardive dyskinesia. Magnetic resonance imaging of the brain was negative. The dyskinetic movements completely resolved in 6 weeks after discontinuation of risperidone. Subsequently, the patient's behavior, sleep, and gait patterns also improved. He was not restarted on risperidone.

The promise of prompt and significant benefit of second-generation antipsychotics to reduce the incidence of tardive dyskinesia has not been borne out in studies.[3] Atypical neuroleptic drugs such as risperidone remain a risk factor for the development of tardive dyskinesia. Patients presenting with gait and postural abnormalities should be evaluated for tardive dyskinesia using validated rating scales such as the Abnormal Involuntary Movement Scale (AIMS)[4] and the Dyskinesia Identification Scale: Condensed User Version (DISCUS)[5] developed for people with developmental disabilities.[6] Unfortunately, these rating scales are rarely used in daily clinical practice.[7] This may be due in part to clinicians' limited knowledge on the use and interpretation of the AIMS and DISCUS.[7] The reported case is a stark reminder that clinicians should use these rating instruments to improve early detection and treatment of potentially reversible cases of tardive dyskinesia.

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