Tardive dyskinesia following risperidone treatment in Tourette's syndrome

Sir,

Tics are involuntary, sudden, repetitive, stereotyped motor movements or phonic productions. Tourette's syndrome (TS) is diagnosed when multiple motor tics and at least one phonic tic is present during the course of the illness. Obsessive-compulsive disorder/symptoms (OCD/OCS) typically manifest as own, egodystonic and involuntary intrusive thoughts, ideas or images accompanied by ritualized, overt, or covert behaviors. The frequent association of TS with obsessive-compulsive symptoms (OCS) and obsessive-compulsive disorder (OCD) is widely recognized.[1] The neurobiology of TS involves frontal subcortical circuits and neurotransmitter systems.[2] The usefulness of antipsychotics in the treatment of TS has been documented, but is often associated with incapacitating side-effects. There are a few case reports of Tardive dyskinesia (TD) in TS after treatment with typical antipsychotics unlike with atypical antipsychotics. We discuss the case of a 24-year-old male who first exhibited repetitive head and neck twisting movements that started at 15 years of age. Nine years after the onset of symptoms when a definitive diagnosis of TS was made, his symptoms included motor and phonic tics [Yale Global Tic Severity Scale (YGTSS) = severe] as well as obsessional thoughts [Yale-Brown Obsessive Compulsive Scale (Y-BOCS) = mild). There was no movement other than tics. His biochemical and neuromaging tests were normal. Initially he was treated with oral sertraline 50 mg and haloperidol 0.5 mg daily, but was lost to follow-up and reported for treatment after a year with identical symptoms. Oral risperidone was started as well as maintained at 1 mg with dinner and fluoxetine was increased to 40 mg with breakfast. Although his tics were minimal at this dose (YGTSS = mild), he developed severe TD of the lower jaw (oro-mandibular dyskinetic movements) after four months of treatment [Abnormal Involuntary Movement Scale (AIMS) rating = severe]. This movement was entirely different from the initial symptoms both anatomically and phenomenologically. Following this, risperidone was stopped and patient was started on vitamin E and clonazepam. In about 45 days there was significant improvement in the dyskinetic movements (AIMS score = mild) but the TS worsened significantly causing severe distress (YGTSS = severe). Risperidone has a higher affinity for 5-hydroxytryptamine 2A receptors and lower dopamine D2 receptor binding than haloperidol.[3] As the 5-HT2A receptor has been implicated in the pathophysiology of TS, risperidone may be of theoretical benefit in TS, especially if the patient has OCD/OCS where 5-HT has been implicated. Several reports illustrate efficacy[4] and lack of extrapyramidal side-effects in TS patients treated with risperidone.[5] To our knowledge, this is the first report of TD complicating the use of risperidone in the treatment of TS. We wish to highlight the need for awareness among clinicians and patients about the potential risk of getting new movement disorders during the course of treatment with risperidone for tic disorders.

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