

Tardive dyskinesia: 5 Steps for prevention

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Tardive dyskinesia (TD) is an elusive-to-treat adverse effect of antipsychotics that has caused extreme discomfort (in a literal and figurative sense) for patients and their psychiatrists. In 2017, the prevalence of TD as a result of exposure to dopamine antagonists was approximately 30% with first-generation antipsychotics and 20% with second-generation antipsychotics.¹ There have been several effective attempts at reducing rates of TD, including lowering the dosing, shifting to second-generation antipsychotics, and using recently introduced pharmacologic treatments for TD. The past 2 years have seen increased efforts at treating this often-irreversible adverse effect with pharmacotherapy, such as the recently marketed vesicular monoamine transporter-2 (VMAT2) inhibitors valbenazine and deutetrabenazine, as well as the supplement Ginkgo biloba,² although issues with cost, adverse effects, or drug–drug interactions could limit the benefits of these agents.

Despite these strategies, one approach has been largely overlooked: prevention. Although it is included in many guidelines and literature reports, prevention has become less of a standard of practice and more of a cliché. Prevention is the key strategy for lowering the rate of TD, and it should be the assumed responsibility of each clinician in every prescription they write throughout the entire continuum of care. Here, we provide steps to take to help prevent TD, and what to consider when TD occurs.

1. Realize that we are all responsible for TD. We know TD exists, but we often feel that this adverse effect is not our fault. Avoid adapting a philosophy of “someone

else caused it,” “they didn’t cause it yet,” or “it’s going to happen anyway.” We must remember that every unnecessary exposure to a dopamine antagonist increases the risk of TD, even if we don’t see the adverse effect firsthand.

2. Treat first-episode psychosis early and aggressively. Doing so may prevent chronicity of the illness, which would save a patient from long-term, high-dose exposure to antipsychotics. Lower the risk of TD with atypical antipsychotics and offer long-acting injectables when possible to improve medication adherence.

3. Treat both acute and chronic symptoms of psychosis throughout the continuum of care. The choice of medication and dose should be reevaluated at each interaction to enhance improvement of acute symptoms and to minimize chronic adverse effects. Always recognize the differences in aggressive treatment of an acute episode of psychosis vs maintenance treatment of baseline symptoms. Also, assess for TD by conducting abnormal involuntary movement scale (AIMS) examinations at baseline and at least biannually.

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4. Use clozapine instead of 2 antipsychotics in chronic, refractory patients when possible. Clozapine is largely underutilized, despite continued evidence of its superiority in effectiveness and prevention of relapse³ vs other agents, and has a lower risk of TD. The use of polypharmacy, on the other hand, has continued to display a lack of added benefit in treating symptoms of psychosis, and an increase in adverse effects.⁴

5. Consider pharmacotherapy if TD has already occurred. Psychiatrists have been waiting for pharmacologic options for treating TD for quite some time. Explore using VMAT2 inhibitors and other agents when it is too late to implement prevention or when a patient's symptoms are refractory to other

treatments. However, avoid anticholinergic medications; there is insufficient data to support the use of these agents in the treatment of TD.⁵

References

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