Current Psychiatry

Pearls

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Make tardive dyskinesia passé with PASST principle

Often disfiguring and irreversible, tardive dyskinesia (TD) remains a problem for patients on maintenance antipsychotics. Central dopamine blockade is believed to contribute to TD's pathology, but the exact cause remains unknown and treatment results are variable.[1,2]

To optimally manage TD, remember the PASST principle—an acronym that includes strategies to prevent, assess, switch, suppress, and treat TD. This principle—based on clinical practice, colleague experiences, and literature reviews—has been helpful for training residents how best to manage this difficult condition.

Prevent. To lower your patient's risk of developing TD:

- reconsider whether an antipsychotic is needed, especially in high-risk patients who are older, have negative symptoms of schizophrenia, experience acute extrapyramidal symptoms, or have affective disorders[3]
- prescribe atypical antipsychotics, which are less likely than the typical agents to produce TD
- use the minimum effective dosage and duration.

Assess. Screen for dyskinetic movements before you start an antipsychotic and approximately every 6 months, using the Abnormal Involuntary Movement Scale (AIMS). The AIMS is easy to administer and score and can detect subtle dyskinesias at an early stage.

Switch. If you identify TD, stop the offending antipsychotic. Switch to a different drug class if psychotic relapse is not an issue (for example, in a patient taking an antipsychotic for treatment-resistant depression).

For patients who require maintenance treatment with antipsychotics, switch from a first-generation antipsychotic to an atypical. Second-generation agents such as olanzapine carry less TD risk than conventionals such as perphenazine. There may be differential risk among atypicals as well (for example, quetiapine is probably less likely to cause TD than risperidone).[1] Also, TD triggered by one atypical may respond to another.[4]

Suppress. It may take time for a medication switch to decrease TD symptoms, if it happens at all. If a patient experiences dangerous or bothersome symptoms such as difficulty breathing or eating, increasing the antipsychotic dosage for a few weeks often provides short-term relief; reserve this approach for urgent clinical situations where switching antipsychotics would take too long or would otherwise be impractical.

Treat. Clozapine is first-line treatment for TD.[5] A variety of non-antipsychotic medications have been used to reduce TD symptoms with inconsistent results (*Table, page 102*). Most carry mild side-effect

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Table

Non-antipsychotic treatments that may reduce TD symptoms

Class	Agent	Comments
Antioxidants	Vitamin E	>1,200 IU/d may be best to prevent deterioration, but cardiac risks increase at dosages >400 IU/d
GABA agonists	Benzodiazepines	Helps some patients, but may have nonspecific sedative effect; abuse potential
	Gabapentin	>1,200 mg/d may help TD and/or blepharospasm
5HT agonists	Buspirone	>120 mg/d
Calcium channel blockers	Verapamil	160 mg/d
Anticholinergics	Benztropine	Worsens TD initially, may help later; recommended for tardive dystonia
Others	Chelated manganese	50 mg/d, especially when combined with vitamin E
	Vitamin B6	300 mg/d
	Melatonin	10 mg/d
	Branched chain amino acid mix (Tarvil)	222 mg/kg tid superior to placebo in one study[6]

risks and could be considered for patients who wish to try something to help alleviate symptoms.

Informed consent and collaborative decision-making are essential to managing TD. Inform patients of TD risk before starting an antipsychotic. If TD occurs, include them in decisions by explaining the risks, benefits, and reasons for switches and treatments. Some patients choose to tolerate mild TD so they can keep taking a medication that helps them stay well. Extensively document these discussions—along with your thought processes—in the medical record.

References

1. Simpson GM. The treatment of tardive dyskinesia and tardive dystonia. *J Clin Psychiatry* 2000;61(Suppl 4):39-44.
2. Kulkarni SK, Naidu PS. Pathophysiology and drug therapy of tardive dyskinesia: Current concepts and future perspectives. *Drugs Today* 2003;39:19-49.
3. Gardos G. Tardive dyskinesia: How to prevent and treat a lingering nemesis. *Current Psychiatry* 2003;2(10):59-66.
4. Suzuki E, Obata M, Yoshida Y, Miyaoka H. Tardive dyskinesia with risperidone and anticholinergics. *Am J Psychiatry* 2002;159(11):1948.
5. Louzã MR, Bassitt DP. Maintenance treatment of severe tardive dyskinesia with clozapine: 5 years' follow-up. *J Clin Psychopharmacol* 2005;25(2):180-2.
6. Richardson MA, Bevans ML, Read LL, et al. Efficacy of the branched-chain amino acids in the treatment of tardive dyskinesia in men. *Am J Psychiatry* 2003;160:1117-24.