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Tardive Dyskinesia

Tardive Dyskinesia (TD) is defined as a persistent, involuntary movement disorder characterized by abnormal, repetitive movements of the face, torso, and extremities. Affecting nearly 1 in 4 individuals prescribed a psychotropic medication, specifically medications that inhibit dopamine (e.g., antipsychotics and antiemetics), TD has the potential to interfere with normal daily activities such as eating, drinking, and walking, as well as significantly impact an individual's mental health.

To emphasize the substantial burden TD has on patients, consider the following statistics:

- An estimated 800,000 people in the U.S. are currently living with TD, with nearly 60% of individuals yet to be officially diagnosed.
- Approximately 30% of patients taking a first-generation antipsychotic develop TD.
- Individuals face an increase of 26.2% in total all-cause healthcare costs post TD diagnosis.
- There is a 49% estimated risk of developing TD for individuals prescribed antipsychotics for 10 or more years.

Identifying TD

As TD is a drug-induced movement disorder frequently associated with medications that block dopamine (e.g., antipsychotics), thorough medication review is a crucial part of the assessment process. Physicians should note that other medications like antiemetics (e.g., metoclopramide, prochlorperazine, and trimethobenzamide) can also cause TD as well. However, unlike other movement disorders such as drug-induced Parkinsonism and acute dystonia, anticholinergic medications may worsen or exacerbate TD symptoms.

Onset of symptoms is another key defining feature of TD. Typically, symptoms begin within 3 months of starting a psychotropic medication but can begin as soon as 1 month in adults 60 years of age and older as they are more sensitive to adverse effects. Changes in dosage or medication discontinuation can also trigger TD development. However, since TD movements are persistent, symptoms will continue beyond 4 weeks of stopping a medication (or beyond 8 weeks for long acting injectables).

Lastly, physicians should periodically observe the patient to assess frequency and severity of movements. TD movements can range from either **athetotic** (slow, writhing) or **choreiform** (rapid, jerking).

TD Symptoms

Facial Movements

- Excessive Blinking
- Grimacing or Frowning
- Grunting
- Puffing Out Cheeks
- Protrusion of the Tongue
- Puckering or Smacking of the Lips

Truncal Movements

- Backwards Arching
- Axial Pulling or Twisting
- Irregular Respirations
- Shoulder Shrugging
- Rocking or Swaying
- Rotating or Thrusting Hips

Extremity Movements

- Hyperextending the Toes
- Knocking Knees
- Tapping of the Feet
- Twisting of the Ankles and Wrists
- Wiggling Fingers ("Piano-Playing Fingers")

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Clinical measurement tools such as the **Abnormal Involuntary Movement Scale (AIMS)**, allow for quick scoring of select target movements to evaluate overall symptom severity.

Other Drug-Induced Movement Disorders

TD is just one of multiple types of drug-induced movement disorders including **Drug-Induced Parkinsonism, Akathisia**, and **Acute Dystonia**. In common, all of these disorders are characterized by involuntary movements associated with medication use. However, multiple differences exists and treatment for each disorder can differ. Review the following table to learn more about the unique characteristics of each movement disorder.

	Tardive Dyskinesia	Drug-Induced Parkinsonism	Akathisia	Acute Dystonia
Onset	Develops over months or years of treatment	Develops within days to months of treatment	Develops within days to months of treatment	Develops within hours to days of treatment
Symptoms	• Movements can be rapid, nonrepetitive, and jerky **OR** slow, continual, and sinuous • Commonly seen in the face, torso, and extremities • Movements may be associated with pain or discomfort	• Bradykinesia, rigidity and tremor • Reduced arm swinging • Shuffling or freezing gait • Masked facial expressions	• Inner restlessness and inability to sit still • Significant distress if unable to move • Tapping of the fingers or toes, continually shifting position, rocking back and forth	• Sustained, involuntary muscle contractions • Mostly confined to jaw, neck, back, and extremities • Painful and/or life-threatening
Movement Type	Athetotic or Choreiform	Bradykinesia	Inner Restlessness	Hypokinetic
Medication Influence	• Anticholinergics may worsen symptoms • Decreasing dose of antipsychotic may precipitate symptoms • Increasing dose of antipsychotic may temporarily improve symptoms	• Anticholinergics may improve symptoms • Decreasing dose of antipsychotic may improve symptoms • Increasing dose of antipsychotic may worsen symptoms	• Anticholinergics may not affect symptoms • Decreasing dose of antipsychotic may improve symptoms • Increasing dose of antipsychotic may worsen symptoms	• Anticholinergics may improve symptoms • Decreasing dose of antipsychotic may improve symptoms • Increasing dose of antipsychotic may worsen symptoms
Reversibility	Potentially reversible; typically persists despite stopping the offending agent	Typically reversible after stopping the offending agent	Typically reversible after stopping the offending agent	Typically reversible after stopping the offending agent

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Approaching TD Treatment

1. **Review the resident's medication regimen.**
   a. What psychotropic medications is the patient currently receiving?
   b. Were any of these medications recently adjusted or discontinued?
   c. Are there other medications potentially causing or worsening the patient's symptoms (e.g., anticholinergics)?

2. **Assess presence and severity of TD symptoms.**
   a. Assess for the development or progression of drug-induced movement disorders, like TD, at baseline and at each follow up encounter.
   b. Ask the patient to elaborate on how their TD symptoms affect functional capability and quality of life.
   c. Utilize clinical measurement tools such as the Abnormal Involuntary Movement Scale (AIMS) to continually track presence and/or progression of movements.

3. **Trial dose reductions and remove offending agents.**
   a. If appropriate, remove offending medications from the patient's medication regimen to reduce severity of symptoms.
   b. For medications that can't be safely discontinued, consider gradually reducing the patient's dose to find the minimum effective dose.

4. **Initiate pharmacologic therapy to treat TD Symptoms.**
   a. Pharmacologic therapy should be reserved for patients unable to discontinue or unable to tolerate psychotropic dose reductions.
   b. For patients with moderate-severe TD associated with antipsychotic therapy, **Vesicular Monoamine Transporter 2 (VMAT2) Inhibitors** are recommended as first-line agents.
      i. VMAT2 inhibitors work by decreasing the uptake of monoamines (e.g., dopamine) reducing the frequency of abnormal, involuntary movements.
   c. There are currently three VMAT2 Inhibitors available in the U.S.
      i. **Valbenazine (Ingrezza)**
         - Offers better adherence due to its once-daily dosing and fewer adverse effects.
         - Available in a sprinkle formulation which can be opened and sprinkled over soft foods for patients with dysphagia.

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ii. Deutetrabenazine (Austedo)

- Immediate and extended-release formulations allow for flexible dosing.
- Associated with lower rates of sedation.

iii. Tetrabenazine (Xenazine)

- Reserved as an alternative agent as it has a higher risk of adverse effects.
- Relatively a low-cost option compared to the other available agents.

d. If therapy with a VMAT2 inhibitor is warranted, physicians should discuss potential adverse effects, boxed warnings and alternative treatments before initiating the selected medication.

Additional Resources

- MIND-TD Questionnaire: Facilitates patient and provider discussion during TD evaluation.
- IMPACT-TD Scale: Estimates the level of impact associated with TD movements.

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