

# Psychiatric Times™ Case-Based Psych Perspectives

## Expert Perspectives on Recognition and Management of Tardive Dyskinesia

Clinical experiences and insights in this *Psychiatric Times*™ Case-Based Psych Perspectives were shared by Leslie L. Citrome, MD, MPH, a psychiatry specialist from Valhalla, New York, and Rose Mary Xavier, PhD, MS, RN, PMHNP-BC, a research scientist and psychiatric nurse practitioner from Chapel Hill, North Carolina. This article provides a summary of key discussion topics from this custom program. Participant bios and financial disclosures are available online.

Xavier highlighted that antipsychotic medications are used for both primary psychotic disorders and mood disorders, including bipolar disorder, and as adjunctive treatment for major depressive disorder. Though the atypical antipsychotics commonly used today carry less risk for tardive dyskinesia (TD) than earlier agents, faculty agreed that the risk of TD remains significant with atypical agents. Citrome said that atypical antipsychotics block postsynaptic dopamine-2 receptors, the putative mechanism underlying the development of TD.

Treating TD is not as simple as stopping the antipsychotic agent, particularly if it is keeping a patient stable. “We need to look at interventions that allow us to continue ongoing treatment for the underlying psychiatric disorder. The option in my mind would be a VMAT2 inhibitor [vesicular monoamine transporter type-2 inhibitor], of which 2 are approved by the FDA for the treatment of tardive dyskinesia, which is added to the patient’s ongoing regimen,” said Citrome.

### Symptom Recognition and Impact on Patients

Faculty cited some of the movements that are characteristic of classic TD: fingers moving as if playing the piano, shoulder shrugging, facial grimacing, tongue protrusion at random times, chewing, and blepharospasm. Oftentimes, symptoms of TD are reported by family members or case managers.

According to Citrome, though patients with mood disorders are often more insightful about abnormal movements, those with schizophrenia can also become aware of these movements. This can lead to heightened anxiety, paranoia, and social isolation. “When we’re trying to transition someone back into the community, it’s really very stigmatizing to have these abnormal movements that set you apart from other people,” he noted. Xavier emphasized the importance of considering the level of distress that patients experience from TD. Citrome agreed, acknowledging that what providers consider to be mild TD can be viewed as severe by the patient and significantly disrupt their ability to function and interact with others.

### Diagnosis of Tardive Dyskinesia

Before starting any antipsychotic (dopamine-2 receptor blocking agent), and every 6 to 12 months

while taking an antipsychotic, the patient should be screened for abnormal movements. “The Abnormal Involuntary Movement Scale [AIMS]...is the gold standard,” said Citrome. He briefly described the AIMS,<sup>1</sup> emphasizing that the tool is very helpful for distinguishing TD symptoms from drug-induced parkinsonism as well as a range of other movement disorders.

Citrome and Xavier agreed that if necessary, the AIMS can reliably be performed via telepsychiatry visits. Citrome noted that recording the video and zooming in can be helpful for confirming abnormal movement. He further added that “it’s helpful to have someone there with the patient, holding the camera, holding the phone from a distance.”

Aside from the AIMS, other clues can help inform the diagnosis of TD. “If [the abnormal movement] emerges a year or 2 [after initiating treatment with a dopamine-2 blocking agent], and they didn’t have any problems before then, we’re going to be very suspicious of tardive dyskinesia,” according to Citrome. He also mentioned an additional clue to correct diagnosis, stating, “If we lower the dose of the antipsychotic, the drug-induced parkinsonism should get better, but the TD may look a little worse.”

### Guideline-Directed Treatment of Tardive Dyskinesia

With their most recent guideline for the treatment of schizophrenia, the American Psychiatric Association recommends the use of VMAT2 inhibitors for treatment of moderate to severe TD.<sup>2</sup> The recommendation extends to patients with any degree of TD if the movements result in functional impairment that the patient or their care providers are aware of.<sup>2</sup> “The level of evidence is pretty strong that VMAT2 inhibitors actually do work well for tardive dyskinesia symptoms,” noted Xavier. Citrome concurred, highlighting that VMAT2 inhibitors produced rapid (within 6 weeks) and durable reductions in TD symptoms in clinical trials.

Both FDA-approved agents, deutetrabenazine and valbenazine, reversibly inhibit VMAT2, a transporter that regulates dopamine uptake from the cytoplasm to the presynaptic vesicle for storage and release into the synapse.<sup>3,4</sup> When that neuron is activated, the vesicles migrate to the presynaptic membrane, where they fuse and deposit the dopamine into the synapse, allowing dopamine to diffuse across the synapse to activate the postsynaptic dopamine-2 receptors. If the VMAT2 transport pump is blocked by the VMAT2 inhibitor, dopamine is depleted from the vesicles, resulting in a decrease in dopamine released into the synapse. This ultimately results in a decrease of dopamine agonism of the postsynaptic dopamine-2 receptors,

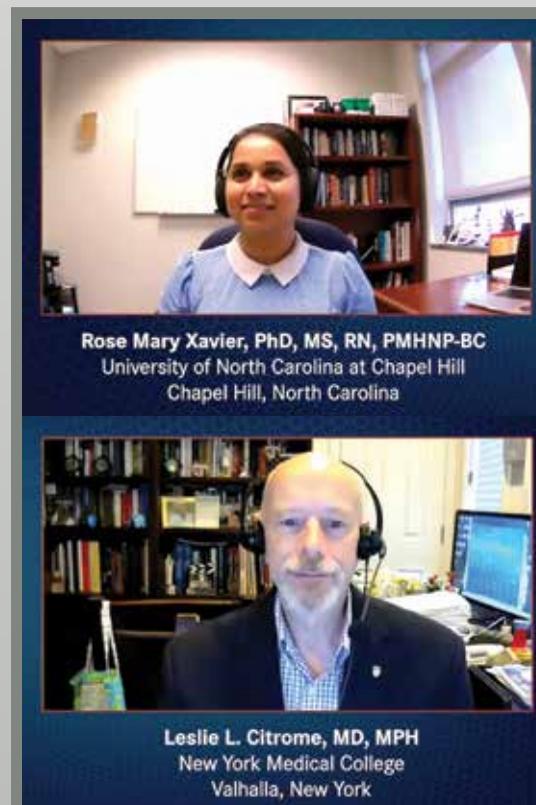
which has the ultimate effect of decreasing the TD movements. According to Xavier, structured and ongoing assessment is important for monitoring response to VMAT2 inhibitor therapy.

### Conclusion

Diagnosis of TD should be based on a formal, structured assessment of abnormal movements, and guidance on whether to treat should stem from how symptoms are affecting the patient. With the advent of VMAT2 inhibitors, “the door is open to the treatment of tardive dyskinesia from mild on up for people who need it, and the treatment guidelines certainly encourage us to do so,” Citrome concluded.

### REFERENCES

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