Clinical Features of Tardive Dyskinesia

[MUSIC PLAYING]

LESLIE LUNDT: Hello, and welcome to a roundtable discussion of the clinical features of tardive dyskinesia. I'm Leslie Lundt, psychiatrist and medical director at Neurocrine Biosciences. I'm joined by Jeremy Schreiber, psychiatric mental-health nurse practitioner at Coleman Professional services; Dr. Carlie Tanner, professor of neurology at the University of California, San Francisco; and Dr. Andrew Cutler, chief medical officer at Meridian Research.

Today, we will highlight basic clinical characteristics of tardive dyskinesia by discussing the condition's phenomenology, potential symptom severity, and clinical course. Before we begin, would you please provide some brief background information about tardive dyskinesia, Dr. Tanner? The term "tardive dyskinesia" refers to abnormal involuntary movements or dyskinesia manifesting in a delayed or tardive manner after prolonged use of Dopamine Receptor Blocking Agents, or DRBAs.

Tardive Dyskinesia, or TD, is defined as involuntary athetoid or choreiform movements. Athetoid movements are slow, sinuous, and continual, whereas choreiform movements are rapid, jerky, and nonrepetitive. By definition, the involuntary movements must develop in association with prolonged exposure to antipsychotics or other DRBAs. As stated, antipsychotics, also known as neuroleptics, are DRBAs. DRBAs are used to manage psychiatric disorders, such as psychosis, depression, and bipolar disorder, as well as gastrointestinal problems.

And when it comes to timing, I think clinicians should know that symptoms may develop after a shorter period of medication use in older patients. In some patients, dyskinesia may arise and/or persist after medication discontinuation, change, or decrease in dose. Another movement disorder called neuroleptic withdrawal-emergent dyskinesia can also arise after change or discontinuation of antipsychotics and usually lasts less than four to eight weeks. Dyskinesia that persists longer than this period is considered tardive dyskinesia. TD movements can involve the tongue, lower face and jaw, and extremities, as well as the pharyngeal, diaphragmatic, or trunk muscles.

Thank you both. That overview of TD is a helpful starting point for our discussion today. Now let's dive a little deeper. How can clinicians recognize TD based on its clinical presentation?

That's a great question because identifying a patient who potentially has TD is the first step in determining an accurate diagnosis. It is important to understand the risk factors for developing TD. The most important risk factor is the prolonged exposure to antipsychotic or other DRBAs. By definition, this is the cause of TD.

Also important is recognizing the clinical characteristics. TD is characterized by involuntary, nonrhythmic, repetitive, purposeless hyperkinetic movements.

What parts of the body are typically affected by TD? As a matter of fact, TD can present essentially in any part of the body, but patients and health-care providers may initially notice facial phenomenology involving the tongue jaw, lips, cheeks, or eyes. Other involuntary movements can include tongue curling, twisting, and protrusion, or lip smacking, licking, puckering, and pursing, or chewing, and lateral jaw movements, or grimacing and bulging of cheeks, or increased blink frequency and tight eyelid closure.

Right. This is consistent with the fact that although TD can present generally in any part of the body, health-care providers are likely more familiar with what they consider to be classic TD, in which the cheeks, mouth, and tongue are mainly involved, otherwise known as the oro-bucco-lingual features.
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