Fast Five Quiz: Assessment of Tardive Dyskinesia

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The accurate and timely assessment of tardive dyskinesia (TD) presents a persistent challenge in clinical practice, particularly given its potential for irreversible motor symptoms and significant impact on patient quality of life. This Fast Five quiz aims to reinforce essential knowledge regarding TD identification, screening protocols, and differentiation from other movement disorders, enabling clinicians to improve early detection and management strategies.

KEY TAKEAWAYS

- The Pivot: Early and accurate identification of TD is critical for mitigating long-term neurological sequelae.
- The Data: The Abnormal Involuntary Movement Scale (AIMS) is a widely validated tool for quantifying TD severity and monitoring treatment response.
- The Action: Clinicians should implement routine, structured screening for TD in patients receiving dopamine receptor blocking agents, even in the absence of overt symptoms.

Background

Tardive dyskinesia (TD) is a hyperkinetic movement disorder characterised by involuntary, repetitive movements, most commonly affecting the orofacial region, but also involving the trunk and extremities. It is primarily associated with long-term use of dopamine receptor blocking agents (DRBAs), including antipsychotics and certain antiemetics. The pathophysiology involves a complex interplay of dopamine receptor hypersensitivity and other neurochemical changes. The prevalence of TD varies, with older antipsychotics carrying a higher risk compared to newer atypical agents. However, even atypical antipsychotics are not without risk, necessitating vigilant monitoring. Early recognition is paramount, as established TD can be challenging to manage and may become irreversible. The clinical dilemma lies in differentiating TD from other drug-induced movement disorders and primary neurological conditions, as well as initiating appropriate interventions. Understanding the underlying mechanisms of DRBA action and their impact on dopamine pathways is crucial for comprehending TD development. DRBAs block dopamine D2 receptors, which can lead to an upregulation or hypersensitivity of these receptors over time, particularly in the striatum. This hypersensitivity is thought to contribute to the involuntary movements characteristic of TD when dopamine transmission is restored or fluctuates. Genetic predispositions and individual patient factors, such as age and comorbidities, also influence susceptibility to TD.

Assessment of Tardive Dyskinesia

Diagnosis of TD is clinical, based on observable involuntary movements and a history of exposure to DRBAs. The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), provides specific criteria for tardive dyskinesia, requiring involuntary movements of at least one month's duration in a patient exposed to a DRBA for at least a

few months, or for a shorter period in older patients. The movements must not be attributable to another medical condition or substance use. The Abnormal Involuntary Movement Scale (AIMS) is the most widely used and validated tool for the assessment of TD. It is a 12-item clinician-rated scale that quantifies the severity of involuntary movements in seven body regions: face, lips, jaw, tongue, upper extremities, lower extremities, and trunk. Each item is scored on a 0-4 scale, where 0 indicates no movements and 4 indicates severe movements. A total score of 2 or more in at least two different body regions, or a score of 3 or 4 in a single body region, is generally considered indicative of TD. Regular AIMS assessments, typically every 3 to 6 months for patients on DRBAs, are recommended to detect emergent TD or monitor progression. Differential diagnosis is crucial. Other conditions that can mimic TD include spontaneous dyskinesias, stereotypies, tics, chorea, dystonia, and akathisia. Drug-induced parkinsonism, another common side effect of DRBAs, presents with bradykinesia, rigidity, and tremor, which are distinct from the hyperkinetic movements of TD. Careful history taking, including medication review and duration of exposure, along with a thorough neurological examination, aids in accurate diagnosis. The emergence of TD often necessitates a review of the patient's medication regimen, including dose reduction, discontinuation of the offending agent if clinically feasible, or switching to an antipsychotic with a lower propensity for TD. Newer pharmacotherapies, such as vesicular monoamine transporter 2 (VMAT2) inhibitors, have demonstrated efficacy in reducing TD symptoms. These inhibitors work by reducing the presynaptic uptake and storage of dopamine, thereby decreasing the amount of dopamine released into the synapse and mitigating the effects of dopamine receptor hypersensitivity. The choice of intervention depends on the severity of TD, the patient's underlying psychiatric condition, and their tolerance to alternative treatments. Patient education regarding the risks of TD and the importance of regular monitoring is also a critical component of management. Furthermore, the impact of TD extends beyond motor symptoms, often leading to significant psychological distress, social stigma, and impaired quality of life for affected individuals. Therefore, a comprehensive management approach includes not only pharmacological interventions but also supportive care and psychological support to address these broader consequences. The long-term prognosis for TD varies, with some cases showing improvement with treatment, while others may persist despite interventions, highlighting the importance of early detection and proactive management strategies.

CLINICAL IMPLICATIONS

The persistent under-recognition of tardive dyskinesia remains a significant concern in general practice and specialist psychiatric care. Despite clear diagnostic criteria and validated assessment tools like the AIMS, routine screening is often inconsistent. This oversight can lead to delayed intervention, potentially allowing symptoms to become entrenched and less responsive to treatment. Clinicians must integrate structured AIMS assessments into their regular follow-up for all patients on dopamine receptor blocking agents, not just those exhibiting overt symptoms. The argument that such assessments are time-consuming pales in comparison to the long-term morbidity associated with unmanaged TD.

The pharmaceutical industry has responded with novel agents, specifically VMAT2 inhibitors, which offer symptomatic relief for established TD. While these represent a welcome addition to the therapeutic armamentarium, their availability should not diminish the emphasis on prevention and early detection. Relying solely on pharmacotherapy for symptomatic TD without robust screening protocols is akin to treating the consequence without addressing the antecedent. Furthermore, the cost-effectiveness of these newer agents must be considered in the broader context of healthcare resource allocation, particularly if earlier detection

> could mitigate the need for intensive pharmacological intervention.
> Ultimately, the onus is on prescribers to balance the therapeutic benefits of DRBAs with the inherent risk of TD. This requires a proactive approach to patient education, ensuring individuals understand the potential for involuntary movements and are empowered to report any emergent symptoms. Guidelines from bodies such as the American Psychiatric Association advocate for regular monitoring, yet adherence varies. A more systematic, perhaps even mandated, approach to TD screening could significantly improve patient outcomes, reducing the burden of a preventable and often irreversible condition.

EDITORIAL & AI STANDARDS

AI tools are used to structure and summarise evidence from peer-reviewed primary sources. Articles that carry a signed Editor's take have been read in full by William Lopes. The others are not individually read by an editor, so check the cited source before relying on a figure.

REFERENCES

1. 1. Kane JM, et al. Revisiting the Abnormal Involuntary Movement Scale: Proceedings From the Tardive Dyskinesia Assessment Workshop. J Clin Psychiatry. 2018;79(3). doi:10.4088/JCP.17cs11959
2. 2. Petriceks A, et al. Assessment and Treatment of Abnormal Involuntary Movements: A Clinically Focused Narrative Review. Harv Rev Psychiatry. 2024;32(2):47-57. doi:10.1097/HRP.0000000000000390
3. 3. Takeuchi H, et al. Pathophysiology, prognosis and treatment of tardive dyskinesia. Ther Adv Psychopharmacol. 2022;12:20451253221117313. doi:10.1177/20451253221117313
4. 4. Carbon M, et al. Tardive Dyskinesia Prevalence in the Period of Second-Generation Antipsychotic Use: A Meta-Analysis. J Clin Psychiatry. 2017;78(3):e264-e278. doi:10.4088/JCP.16r10832
5. 5. Woods SW, et al. Incidence of tardive dyskinesia with atypical versus conventional antipsychotic medications: a prospective cohort study. J Clin Psychiatry. 2010;71(4):463-74. doi:10.4088/JCP.07m03890yel
6. 6. Carbon M, et al. Tardive dyskinesia risk with first- and second-generation antipsychotics in comparative randomized controlled trials: a meta-analysis. World Psychiatry. 2018;17(3):330-340. doi:10.1002/wps.20579
7. 7. O'Brien A. Comparing the risk of tardive dyskinesia in older adults with first-generation and second-generation antipsychotics: a systematic review and meta-analysis. Int J Geriatr Psychiatry. 2016;31(7):683-93. doi:10.1002/gps.4399
8. 8. Tarsy D, et al. Epidemiology of tardive dyskinesia: is risk declining with modern antipsychotics?. Mov Disord. 2006;21(5):589-98. doi:10.1002/mds.20823

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