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Non-Medication Induced Tardive Dyskinesia

Thank you for contacting Neurocrine Biosciences with your unsolicited Medical Information request regarding the cause of non-medication induced tardive dyskinesia.

The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision (DSM-5-TR) defines the essential features of tardive dyskinesia as abnormal, involuntary movements of the tongue, jaw, trunk, or extremities that develop in association with the use of medications that block postsynaptic dopamine receptors, such as first- and second-generation antipsychotic medications and other medications such as metoclopramide for gastrointestinal disorders.[1] The movements are present over a period of at least 4 weeks and may be choreiform (rapid, jerky, nonrepetitive), athetoid (slow, sinuous, continual), or semirhythmic (e.g., stereotypies) in nature; however, the movements are distinctly different from the rhythmic (3-6 Hz) tremors commonly seen in medication-induced parkinsonism.[2] Although a large number of epidemiological studies have established the etiological relationship between dopamine blocking drug use and tardive dyskinesia, any dyskinesia in an individual who is receiving antipsychotic medication is not necessarily tardive dyskinesia.[7]

The symptoms of tardive dyskinesia tend to be worsened by stimulants, antipsychotic medication withdrawal, and anticholinergic medications (such as benztropine, commonly used to manage medication-induced parkinsonism) and may be transiently worsened by emotional arousal, stress, and distraction during voluntary movements in unaffected parts of the body.[3]

There is no obvious gender difference in the susceptibility to tardive dyskinesia, although the risk may be somewhat greater in postmenopausal women. Greater cumulative amounts of antipsychotic medications and early development of acute extrapyramidal side effects (such as medication-induced parkinsonism) are two of the most consistent risk factors for tardive dyskinesia.[4] Mood disorders (especially major depressive disorder), neurological conditions, and alcohol use disorder have also been found to be risk factors in some groups of individuals.[5]

Dyskinesia that emerges during withdrawal from an antipsychotic medication or other dopamine receptor blocking agent may remit with continued withdrawal from the medication. If the dyskinesia persists for at least 4 weeks, a diagnosis of tardive dyskinesia may be warranted. Tardive dyskinesia must be distinguished from other causes of orofacial and body dyskinesia. These conditions include Huntington's disease, Wilson's disease, Sydenham's (rheumatic) chorea, systemic lupus erythematosus, thyrotoxicosis, heavy metal poisoning, ill-fitting dentures, dyskinesia due to other medications such as l-dopa or bromocriptine, and spontaneous dyskinesias. Factors that may be helpful in making the distinction are evidence that the symptoms preceded the exposure to the antipsychotic medication or other dopamine receptor blocking agent or that other focal neurological signs are present. It should be noted that other movement disorders may coexist with tardive dyskinesia. Because spontaneous dyskinesia can occur in more than 5% of individuals and is also more common in elderly persons, it may be difficult to prove that antipsychotic medications produced tardive dyskinesia in a given individual. Tardive dyskinesia must be distinguished from symptoms that are due to a medication-induced acute movement disorder (e.g., medication-induced parkinsonism, acute dystonia, acute akathisia). Acute dystonia and acute akathisia can develop quickly within hours to days, and medication-induced parkinsonism develops within weeks of initiating or increasing the dose of an antipsychotic medication or other dopamine receptor blocking agent (or reducing the dose of a medication used to treat the acute extrapyramidal symptoms).[6]

This letter is provided in response to your unsolicited medical information inquiry. Please feel free to contact Neurocrine Medical Information at (877) 641-3461 or medinfo@neurocrine.com if you would like to request additional information.

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References:

1. Caroff SN, Ungvari GS, Cunningham Owens DG: Historical perspectives on tardive dyskinesia. J Neurol Sci 389:4–9, 2018
2. Ward KM, Citrome L: Antipsychotic-related movement disorders: drug-induced parkinsonism vs. tardive dyskinesia: key differences in pathophysiology and clinical management. Neurol Ther 7(2):233–248, 2018
3. Citrome L: Clinical management of tardive dyskinesia: five steps to success. J Neurol Sci 383:199–204, 2017
4. Patterson-Lomba O, Ayyagari R, Carroll B: Risk assessment and prediction of TD incidence in psychiatric patients taking concomitant antipsychotics: a retrospective data analysis. BMC Neurol 19(1):174, 2019
5. D'Abreu A, Akbar U, Friedman JH: Tardive dyskinesia: epidemiology. J Neurol Sci 389:17–20, 2018
6. Ward KM, Citrome L: Antipsychotic-related movement disorders: drug-induced parkinsonism vs. tardive dyskinesia: key differences in pathophysiology and clinical management. Neurol Ther 7(2):233–248, 2018
7. American Psychiatric Association. (2022). Diagnostic and statistical manual of mental disorders (5th ed., text rev.). https://doi.org/10.1176/appi.books.9780890425787