



LETTER TO THE EDITOR

Drug-induced stuttering associated with venlafaxine-olanzapine combination: A rare pharmacodynamic interaction

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Dear Editor,

Stuttering is a speech disorder characterized by disruptions in fluency and timing due to the repetition of sounds, syllables, or words. Although it is generally a developmental condition, it can also occur due to neurological or psychogenic causes, or as a rare adverse drug reaction (ADR) associated with various pharmacological agents. Stuttering has been particularly associated with hyperactivity of the dopaminergic system. Therefore, certain drugs such as antidepressants, antiepileptics, antipsychotics, and psychostimulants may contribute to the development of stuttering or trigger this condition (1–4). Among these, atypical antipsychotics and serotonin-norepinephrine reuptake inhibitors (SNRIs) have occasionally been linked to speech disturbances (3). We present a rare case of drug-induced stuttering following the combination of venlafaxine and olanzapine.

A 35-year-old male was admitted to our psychiatric unit after an acute suicide attempt. He was conscious, oriented, and cooperative, with a depressed affect and thought content dominated by family and marital stressors. No psychotic symptoms were observed, though passive suicidal and homicidal ideations were noted. Routine investigations, including bloodwork, inflammatory markers, urine toxicology, and cranial/cervical computed tomography (CT) imaging, were

unremarkable. Psychodynamic evaluation revealed low stress tolerance and impaired impulse control. A diagnosis of major depressive disorder was made (5). Depressive symptoms had started approximately one month earlier, and this was the patient's second depressive episode.

Venlafaxine 37.5 mg/day was initiated and increased to 75 mg/day on day 3. Olanzapine 5 mg/day was added the same day. On the seventh day of combination therapy, the patient developed stuttering symptoms characterized by difficulty initiating speech, sound repetition, and anxiety. The patient had no personal or family history of stuttering, and there was no history of attention-deficit/hyperactivity disorder (ADHD) or tic disorders. The possibility of psychogenic stuttering was excluded based on detailed psychiatric and neurological evaluations. Neurological examination and vital signs were unremarkable. Given a suspected ADR, venlafaxine was discontinued. Further tests, including electroencephalography (EEG), cranial CT, diffusion magnetic resonance imaging (MRI), and magnetic resonance (MR) angiography, were all normal. Ear, nose, and throat (ENT) consultation showed normal findings. The Naranjo ADR Probability Scale score was 6 (probable ADR). As symptoms persisted, olanzapine was discontinued on day 16. By day 23 (seven days after stopping olanzapine), speech returned to normal. Mirtazapine 15 mg/day was initiated as an alternative.

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The neurobiology of drug-induced stuttering is not fully understood. Hypothesized mechanisms include dopaminergic overactivity, reduced gamma-aminobutyric acid (GABAergic) inhibition, anticholinergic effects, and serotonergic dysregulation (1). While olanzapine has been implicated in inducing stuttering (4, 6), it has also been reported as beneficial in rare cases (7). This paradox may reflect individual differences in dopaminergic-cholinergic balance (3).

In our case, stuttering followed venlafaxine dose escalation and olanzapine initiation. Venlafaxine enhances serotonergic tone and may indirectly affect dopamine pathways. Olanzapine acts on dopamine and serotonin receptors and has anticholinergic properties (1, 3). Their combination may result in a pharmacodynamic interaction impairing speech fluency. Some reviews also suggest that serotonergic agents may influence basal ganglia circuits involved in speech (1). Serotonergic drugs disrupt the balance of dopamine and glutamate in the basal ganglia, impairing motor control. This disruption can interrupt the flow of speech and cause stuttering (3).

Only one prior case describes a similar phenomenon (6): stuttering began four days after olanzapine (10 mg/day) was added to ongoing venlafaxine (150 mg/day), resolving two days after stopping olanzapine. In our case, stuttering began on day 10 and resolved seven days after stopping olanzapine, despite venlafaxine already being withdrawn. These cases support a potential interaction between venlafaxine and olanzapine in the pathogenesis of stuttering.

Following symptom resolution, mirtazapine was initiated at 15 mg/day and increased to 30 mg/day on day 3. At the one-month follow-up, the dose was increased to 45 mg/day due to residual depressive symptoms, resulting in clinical improvement. Mirtazapine has not been associated with stuttering and was well tolerated. Written informed consent was obtained from the patient for the publication of this case report.

In conclusion, clinicians should consider ADRs in new-onset stuttering, especially when multiple psychotropics are used. Focusing on the rare occurrence of stuttering induced by the combination of olanzapine and venlafaxine, this case emphasizes the importance of close patient monitoring and step-by-step treatment planning to manage unexpected side effects efficiently. Early recognition may prevent unnecessary neurological evaluations and guide treatment adjustments.

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