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LETTER TO THE EDITOR

Brain zaps after antidepressant discontinuation: Heterogeneous responses across paroxetine, venlafaxine, and duloxetine—a three-case letter

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Dear Editor,

Antidepressant discontinuation syndrome encompasses a constellation of symptoms that may occur following the abrupt cessation or rapid dose reduction of serotonergic reuptake inhibitors, including selective serotonin reuptake inhibitors (SSRIs) and serotonin–norepinephrine reuptake inhibitors (SNRIs) (1). Among these, "brain zaps" are particularly distinctive sensory phenomena, described by patients as sudden, electric shock-like sensations in the head, often triggered by head movements and capable of significantly impairing daily functioning (2). Prevalence estimates vary; recent meta-analyses suggest that approximately 15% of users experience general discontinuation symptoms (3). However, reliance on randomized controlled trial (RCT) samples may limit real-world generalizability and potentially underestimate prevalence in clinical settings (4). Key risk factors include the use of short half-life medications, such as paroxetine and venlafaxine, whereas fluoxetine is associated with a lower risk due to its prolonged elimination profile (5).

The first case involves a 35-year-old male with generalized anxiety disorder who had been treated with venlafaxine 150 mg/day for three years. This was his first attempt to taper the medication. The dose was gradually reduced over three months (150 mg/day to 112.5 mg/day, then to 75 mg/day). Two days after the final reduction to 37.5 mg/day in the third month, he developed electric shock-like sensations triggered by head movements, which he described as "an electrical cable snapping." These episodes lasted 1–3 seconds, occurred 25–30 times daily, and were accompanied by mild imbalance and nausea. Neurological examination revealed no focal deficits. An initial trial of fluoxetine 20 mg/day for four weeks yielded no improvement. Subsequently, short-term diazepam 5 mg/day was added, resulting in a marked reduction in symptoms within one week. Venlafaxine was discontinued in the second week, fluoxetine in the third, and diazepam in the fourth, with no recurrence at two-month follow-up. The Naranjo score was 4 (possible), and the presentation was consistent with acute withdrawal according to Chouinard's classification.

The second case is a 40-year-old female with major depressive disorder and comorbid anxiety who had received paroxetine 30 mg/day for five years. She had no prior discontinuation attempts. The dose was tapered from 30 mg/day to 20 mg/day in the first month. Upon reduction to 10 mg/day in the second month, she experienced electric sensations triggered by head movements, described as "lightning striking in my brain." These episodes lasted 1–2 seconds, occurred 15–20 times daily, and were accompanied by mild imbalance and transient

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Table 1: Total Naranjo Adverse Drug Reaction Probability Scale scores

Case	Drug	Naranjo score	Probability classification
Case 1	Venlafaxine	4	Possible
Case 2	Paroxetine	7	Probable
Case 3	Duloxetine	6	Probable

Adapted from Naranjo et al., Clin Pharmacol Ther. 1981;30(2):239–245 (8).

visual blurring. Neurological examination was unremarkable. Following complete discontinuation of paroxetine, fluoxetine 20 mg/day was initiated. She demonstrated significant improvement by the second week and achieved complete resolution by the fifth week. Fluoxetine was then gradually tapered and discontinued, with no recurrence at three-month follow-up. The Naranjo score was 7 (probable), and the presentation was consistent with acute withdrawal.

The third case concerns a 50-year-old male with generalized anxiety disorder who had been treated with duloxetine for 10 years (60 mg/day for the last five years). A previous tapering attempt two years earlier had failed due to electric shock sensations. Following clinical improvement, a dose reduction from 60 mg/day to 30 mg/day was attempted in the first month. On the second day, he developed electric shock-like sensations triggered by head movements, described as "an electrical current flowing through my brain." These episodes lasted 2–3 seconds, occurred 20–25 times daily, and were accompanied by mild imbalance. Neurological examination was normal. After one week of persistent symptoms, fluoxetine 20 mg/day was initiated. Symptoms persisted for another week before gradually resolving by the third week. Duloxetine was discontinued in the second week, and fluoxetine was continued for an additional two weeks before tapering. One-month follow-up revealed no recurrence. The Naranjo score was 6 (probable), consistent with acute withdrawal.

This three-case series demonstrates heterogeneous treatment responses across different antidepressants. Paroxetine showed a favorable response to fluoxetine bridging, venlafaxine was resistant to fluoxetine but responded to short-term diazepam, and duloxetine exhibited a delayed yet complete response to fluoxetine. These findings suggest that individualized treatment approaches may be necessary. Short-term benzodiazepines may be considered in selected resistant cases, with careful monitoring for risks such as dependence, sedation, and cognitive impairment. The observed variability in treatment response may reflect differences in pharmacodynamic profiles beyond pharmacokinetic properties. Paroxetine's selective serotonergic action responds predictably to serotonergic substitution, whereas venlafaxine's dual serotonergic-noradrenergic effects may render purely serotonergic substitution insufficient (6). For SNRIs, serotonergic substitution alone may inadequately address noradrenergic withdrawal components, potentially necessitating longer bridging periods, as observed with duloxetine (7). The limitations of this series include its small sample size, absence of placebo controls or rechallenge protocols, lack of biomarkers, and Naranjo scores within the "possible" to "probable" range (Table 1). Although the Naranjo scale is designed for adverse drug reactions, it was applied here in the absence of specific discontinuation assessment tools, warranting cautious interpretation. Furthermore, the absence of data on individual metabolic or genetic differences limits the generalizability of these findings. Future research should focus on larger, controlled studies to establish optimal management strategies for this clinically challenging phenomenon.

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