

Gradual Discontinuation of Citalopram After Decades of Maintenance Therapy: A Case Report

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Abstract Long-term antidepressant therapy may present challenges when discontinuation is attempted, particularly after prolonged exposure and previous withdrawal symptoms. We report a woman in her 60s with a history of depression, post-traumatic stress disorder, and generalized anxiety disorder who had taken citalopram 40 mg daily for approximately 23 years and wished to discontinue treatment because of concerns about long-term medication exposure and possible worsening of gastrointestinal symptoms. A gradual, symptom-guided taper was undertaken over approximately nine months from 40 mg to 35 mg, 30 mg, 20 mg, 15 mg, 10 mg, 5 mg, 2.5 mg, and ultimately 0 mg. Early reductions were associated with irritability, insomnia, anxiety, and transient somatic symptoms, with the most prominent symptom cluster occurring after reduction from 30 mg to 20 mg. Extended periods of dose stabilization were used before further reductions. In contrast, later reductions from 10 mg to 5 mg and from 5 mg to 2.5 mg were comparatively well tolerated. A brief episode of passive suicidal ideation while maintained on 2.5 mg prompted additional observation before complete discontinuation. Following cessation, the patient remained off citalopram across two follow-up visits, reported stable mood, and did not experience the anxiety, irritability, or jaw tightness that had previously occurred with delayed or missed doses. This case illustrates that discontinuation after decades of citalopram therapy can be achieved through gradual dose reduction, individualized stabilization periods, and longitudinal clinical monitoring.

Keywords: citalopram, antidepressant discontinuation, antidepressant withdrawal, selective serotonin reuptake inhibitor, deprescribing, tapering, long-term therapy

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1. Introduction

Selective serotonin reuptake inhibitors (SSRIs) are widely prescribed for depressive and anxiety disorders and may be continued for extended periods as maintenance therapy. Long-term antidepressant use is common in clinical practice; in one longitudinal primary care cohort, a substantial proportion of patients receiving antidepressants continued treatment for several years [1]. Although maintenance therapy may be appropriate for patients at risk of recurrence, prolonged treatment can create challenges when discontinuation is later considered.

Reduction or discontinuation of antidepressants can produce a heterogeneous syndrome involving affective, sleep-related, gastrointestinal, sensory, and other somatic symptoms [2,3]. These manifestations may occur after abrupt cessation or dose reduction and can overlap clinically with recurrence of the underlying psychiatric disorder, complicating interpretation during a taper [2,4]. Longer treatment duration and a history of previous withdrawal symptoms may increase vulnerability during discontinuation, although evidence involving patients

treated continuously for decades remains limited [3,4].

Gradual and individualized dose reduction is therefore an important consideration when discontinuing long-term SSRI therapy. Proposed tapering approaches emphasize adjustment of the rate and magnitude of dose reductions according to emerging symptoms rather than adherence to a rigid schedule [4,5]. This report describes the gradual discontinuation of citalopram after approximately 23 years of maintenance therapy. The clinical course was notable for transient affective, sleep-related, and somatic symptoms during several earlier dose reductions, followed by comparatively favorable tolerability during subsequent lower-dose reductions and eventual discontinuation.

2. Case Presentation

A woman in her 60s with a history of depression, post-traumatic stress disorder (PTSD), generalized anxiety disorder (GAD), and alcohol use disorder in sustained remission presented with the goal of discontinuing long-term citalopram therapy. She had taken citalopram 40 mg daily for approximately 23 years and had largely received medication refills through primary care after discontinuing

regular psychiatric follow-up. At presentation, she was psychiatrically stable overall but was not asymptomatic; she continued to report anxiety related to financial and trauma-related stressors, chronic insomnia, and intermittent mood symptoms, without acute safety concerns. She remained engaged in psychotherapy, including eye movement desensitization and reprocessing (EMDR) therapy. She also reported previous discontinuation symptoms when refills were delayed or doses were missed, including increased anxiety, irritability, and jaw tightness. Her desire to taper was driven by concerns about long-term medication exposure and the possibility that citalopram could be contributing to ongoing gastrointestinal symptoms, including irritable bowel syndrome (IBS).

Citalopram was initially reduced from 40 mg to 35 mg daily. She subsequently reported mild irritability, edginess, and insomnia, although these symptoms were less severe than those previously experienced after missed doses. After reaching 30 mg, she continued to experience edginess and intermittent negative thoughts that remained manageable, while insomnia became more prominent with delayed sleep onset, fragmented sleep, and daytime fatigue.

The most noticeable symptom cluster occurred after reduction from 30 mg to 20 mg. Approximately six days after the reduction, she reported increased anxiety, a “quivery” sensation resembling nervousness, increased appetite, and nonspecific feverish and achy sensations. The dose was therefore maintained at 20 mg rather than reduced further. Over subsequent follow-up, these symptoms subsided and emotional stability improved. A brief trial of gabapentin for insomnia was limited by excessive daytime sedation.

After several months of stability at 20 mg, tapering resumed through 15 mg and then 10 mg daily. These later reductions were better tolerated. At 10 mg, she reported improvement in anxiety and irritability and felt that she was managing her depressive symptoms effectively. Citalopram was subsequently reduced to 5 mg without significant difficulty and without recurrence of the earlier edginess.

The dose was then reduced from 5 mg to 2.5 mg daily. She initially remained stable, with only occasional irritability that she attributed to baseline temperament or situational stress. While maintained on 2.5 mg, she later experienced one day of fleeting passive suicidal thoughts followed by approximately two days of feeling unusually low. Because the relationship of these symptoms to the taper, the underlying mood disorder, or situational factors was uncertain, citalopram was maintained at 2.5 mg rather than immediately discontinued. At subsequent follow-up, she again reported doing well overall, with no further passive suicidal thoughts documented.

After several additional weeks at 2.5 mg without recurrent withdrawal symptoms, citalopram was discontinued. At the first post-discontinuation follow-up, she reported doing well and denied withdrawal symptoms. Sleep was less fragmented, although total sleep duration remained approximately 4 to 6 hours nightly and was not fully restorative.

At a second follow-up approximately two weeks after discontinuation, she remained off citalopram and reported

feeling well with stable mood. She specifically denied recurrence of the anxiety, irritability, or jaw tightness that had previously occurred with delayed or missed doses and reported no recent passive death wishes. She continued to experience situational anxiety and sleep deprivation related to ongoing life stressors, but these were not accompanied by her previous discontinuation pattern. She remained off citalopram without medication reinstatement. The patient provided consent for publication of her de-identified clinical course. The overall tapering course is summarized in [Table 1](#).

Table 1. Clinical Course During Gradual Citalopram Discontinuation

Approximate Period	Citalopram Dose	Clinical Course	Management
November to December 2025	40 to 35 mg	Mild irritability, edginess, and insomnia; symptoms less severe than after previously missed doses	Maintained at 35 mg before further reduction
December 2025 to January 2026	35 to 30 mg	Edginess, manageable negative thoughts, prominent insomnia, and daytime fatigue	Continued gradual taper with monitoring
January to February 2026	30 to 20 mg	Increased anxiety, “quivery” sensation, increased appetite, and feverish and achy sensations	Held at 20 mg until symptoms subsided
February to April 2026	20 mg	Progressive emotional stabilization; sleep disturbance gradually improved	Extended stabilization period before further taper
April to May 2026	20 to 15 to 10 mg	Better tolerated; anxiety and irritability improved	Taper resumed
June 2026	10 to 5 mg	No significant difficulty; earlier edginess did not recur	Continued taper
July to August 2026	5 to 2.5 mg	Initially well tolerated; later brief passive suicidal thoughts and several days of low mood	Maintained at 2.5 mg for additional observation
September 2026	2.5 to 0 mg	No characteristic withdrawal symptoms after discontinuation	Citalopram discontinued
1 to 2 weeks after discontinuation	0 mg	Stable mood; no recurrent anxiety, irritability, jaw tightness, or passive death wishes	Remained off citalopram without reinstatement

3. Discussion

This case illustrates the challenges of discontinuing long-term antidepressant therapy after more than two decades of continuous citalopram exposure. The patient entered the taper with a history of recognizable symptoms when doses were delayed or missed and subsequently developed irritability, insomnia, anxiety, and transient somatic symptoms during several early dose reductions. Rather than following a fixed tapering schedule, further dose reductions were delayed when symptoms emerged

and resumed after clinical stabilization. This symptom-guided approach is consistent with literature emphasizing treatment duration, previous withdrawal experiences, and individual tolerability when planning antidepressant discontinuation [3,4,5].

The pharmacology of citalopram provides an important framework for understanding antidepressant tapering. Positron emission tomography studies have shown that conventional therapeutic doses of citalopram produce high serotonin transporter (SERT) occupancy, while the relationship between dose and transporter occupancy is nonlinear rather than proportional [6,7]. At higher doses, increases in dose produce relatively small additional changes in SERT occupancy, whereas reductions at lower doses may produce proportionally larger pharmacodynamic changes. This hyperbolic dose-occupancy relationship provides the mechanistic rationale for progressively smaller dose reductions near the end of an antidepressant taper [5,6,7].

Interestingly, this patient's clinical course did not follow the pattern that might be anticipated from SERT occupancy alone. Her most prominent symptom cluster occurred after reduction from 30 mg to 20 mg, whereas subsequent reductions from 10 mg to 5 mg and from 5 mg to 2.5 mg were comparatively well tolerated. This observation does not contradict the pharmacologic basis of hyperbolic tapering, because transporter occupancy represents only one determinant of clinical response. The interval between dose reductions, adaptation during prolonged dose holds, concurrent sleep disturbance and psychosocial stressors, prior withdrawal sensitivity, and individual susceptibility may all influence tolerability. Thus, pharmacologic models may guide taper design but may not reliably predict which dose reduction will be most difficult for a given patient [7].

Distinguishing antidepressant withdrawal from recurrence of the underlying psychiatric disorder was another important consideration in this case. Irritability, anxiety, insomnia, low mood, and negative thoughts may occur in either setting, making causal attribution difficult on the basis of symptoms alone [4,5]. This uncertainty became particularly relevant when the patient experienced a brief episode of passive suicidal ideation and low mood while maintained on citalopram 2.5 mg. Rather than attributing these symptoms automatically to withdrawal or depressive recurrence, the dose was maintained and the patient was observed longitudinally. After eventual discontinuation, she remained off citalopram across two follow-up visits, reported stable mood, and did not experience the anxiety, irritability, or jaw tightness that had characterized previous missed doses.

Several limitations should be considered. This is a single clinical case and cannot establish an optimal tapering schedule or a causal relationship between individual dose reductions and reported symptoms. Symptoms were assessed during routine clinical care

rather than with a standardized antidepressant withdrawal instrument, and no pharmacokinetic measurements or serotonin transporter imaging were obtained. Concurrent psychotherapy, behavioral sleep interventions, nonprescription supplements, situational stressors, and changes in sleep may also have influenced the observed course. In addition, post-discontinuation follow-up was limited to approximately two weeks, allowing assessment of the immediate outcome but not the long-term risk of depressive or anxiety recurrence. Nevertheless, this case demonstrates that complete discontinuation after more than two decades of citalopram therapy was achievable through a gradual, symptom-guided approach with stabilization periods when clinically necessary.

4. Conclusion

Gradual, symptom-guided tapering allowed successful discontinuation of citalopram after approximately 23 years of continuous use. The patient experienced greater difficulty during earlier dose reductions than at lower doses, underscoring that individual tolerability may not correspond directly to pharmacologic dose-response models. Extended stabilization periods and close clinical monitoring may facilitate antidepressant discontinuation after prolonged treatment.

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