Case Report

Case Series: Marked Improvement in Treatment-Resistant Obsessive–Compulsive Symptoms with Over-the-Counter Glutamatergic Augmentation in Routine Clinical Practice

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Case Series: Marked Improvement in Treatment-Resistant Obsessive–Compulsive Symptoms with Over-the-Counter Glutamatergic Augmentation in Routine Clinical Practice

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Abstract

Background: A sizeable minority of people with obsessive–compulsive disorder (OCD) remain haunted by intrusive thoughts and exhausting rituals even after lengthy courses of high-dose selective serotonin reuptake inhibitors (SSRIs) and antipsychotic add-ons. Because mounting basic-science and imaging data point toward glutamatergic imbalance in OCD, clinicians have begun to explore medicines that target glutamate rather than serotonin alone. Case series: Four consecutive patients—three adults and one 15-year-old—were seen in a community outpatient practice after standard treatments had failed to ease their severe OCD. Each patient was taking (or was started on) fluoxetine 10–40 mg/day, chosen both for mood stabilisation and for its ability to slow CYP2D6 metabolism. Over-the-counter dextromethorphan was then introduced at 30–120 mg/day, divided two or three times daily, making use of the drug's mild NMDA-receptor blockade. Two patients who continued to report mental fog and vivid intrusive images later received piracetam 1 200 mg/day for its positive allosteric effect on AMPA receptors. Follow-up occurred during routine office visits; no research-specific procedures were added. Results: Within two to four weeks of starting dextromethorphan, all four patients described fewer and less intense obsessions, longer delays before engaging in compulsions, and improved sleep. The two individuals given piracetam reported additional gains in mental clarity and a further drop in obsessive content that persisted for at least eight weeks. Side-effects were mild: one brief episode of tachycardia that resolved after a small SSRI dose reduction, mild tremor in two patients, and transient menstrual spotting in one. No dissociative or psychotic reactions were observed. Conclusion: In this small, naturalistic series, adding low-cost glutamatergic agents—dextromethorphan with or without piracetam—to ongoing fluoxetine was linked to meaningful symptom relief in otherwise treatment-resistant OCD.

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Background

Growing evidence places glutamatergic dysfunction—rather than purely serotonergic abnormalities—at the centre of obsessive-compulsive disorder (OCD). Magnetic resonance spectroscopy and cerebrospinal-fluid studies repeatedly show elevated glutamate or Glx in key nodes of the cortico-striato-thalamo-cortical (CSTC) circuit, particularly the anterior cingulate cortex and striatum, while genetic association studies implicate transporters and receptor subunits that regulate synaptic glutamate tone [1,2].

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Within this framework, N-methyl-D-aspartate (NMDA) receptors assume a pivotal role. Because NMDA channels require both ligand binding and postsynaptic depolarisation, they act as coincidence detectors that stabilise synaptic change through calcium-dependent plasticity. Hyperactivation—whether driven by increased glutamate release, impaired re-uptake (e.g., SLC1A1 variants), or gain-of-function in receptor subunits such as GRIN2B—may lock CSTC ensembles into maladaptive high-firing "attractor" states, experienced clinically as obsessions and urges to ritualise [2,3]. Pre-clinical support comes from Sapap3-knockout mice, in which loss of a postsynaptic scaffold protein distorts NMDA currents and produces compulsive grooming that subsides when NMDA signalling is normalised [4].

NMDA pathology rarely occurs in isolation, however, because channel opening normally depends on earlier depolarisation through α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptors. Disturbances that blunt AMPA trafficking or conductance can therefore secondarily damp or dys-time NMDA activity, while excessive AMPA drive can precipitate NMDA-mediated excitotoxic cascades. In Sapap3 mutants, for instance, both AMPA and NMDA receptor localisation is disrupted at striatal synapses, shifting excitation–inhibition balance toward perseverative motor output [4]. Clinically, the rapid anti-obsessional effects of ketamine are believed to start with brief NMDA blockade that paradoxically boosts cortical glutamate release, followed by sustained AMPA-dependent synaptogenesis that may "reset" pathological circuitry [5].

The same glutamate "see-saw" that lifts mood also seems to quiet obsessions and rituals: blocking NMDA receptors loosens the rigid firing patterns in cortico-striato-thalamo-cortical loops, while strengthening AMPA transmission helps the circuit resettle into a calmer, goal-directed rhythm [5]. IV ketamine and add-on memantine already show that nudging this balance can cut Y-BOCS scores, but their cost, logistics, and side-effects limit everyday use [6]. Our oral protocol demonstrated in the following case series tries to reproduce the same neurochemical choreography with inexpensive pharmacy staples. Dextromethorphan (DXM) delivers the brief NMDA "pause" that unleashes a corrective glutamate burst; pairing it with a potent CYP2D6 inhibitor such as fluoxetine or paroxetine keeps DXM in the therapeutic window for hours instead of minutes [7]. For patients whose symptoms rebound quickly—or who need a faster lift—we layer in piracetam. As a mild, well-tolerated AMPA positive modulator, piracetam widens the postsynaptic gate through which that glutamate pulse flows, amplifying BDNF- and mTOR-driven synaptic rewiring that pre-clinical work links to both antidepressant and anti-compulsive effects [8,9].

Methods

This report reviews four back-to-back cases of trauma-related illness managed with an oral glutamatergic add-on protocol at a private outpatient clinic (Tsim Sha Tsui H Zentre Clinic, Chueng Ngo Medical) between December 2024 and November 2025. Every patient was personally assessed and treated by the author.

The core schedule used dextromethorphan hydrobromide 15 mg tablets (usual total dose 30–90 mg/day in divided doses) whose exposure was prolonged by a potent CYP2D6 blocker—either fluoxetine 10–20 mg/day or bupropion XL 150 mg/day. Piracetam 600–1 200 mg/day could be added as an AMPA-positive allosteric modulator. Doses were titrated case-by-case for benefit and tolerability. Pre-existing psychotropics were left in place unless patients chose otherwise. Follow-up visits occurred every 2–8 weeks, combining PHQ-9 and GAD-7 self-ratings with an in-depth clinical interview; formal OCD or PTSD instruments were not used in routine care.

For context, clinic dispensing of the two main agents over the most recent three months (September–November 2025) was:

- September 2025 – 16 448 tablets dextromethorphan 15 mg; 2 595 tablets piracetam 1 200 mg
- October 2025 – 16 940 tablets dextromethorphan 15 mg; 3 485 tablets piracetam 1 200 mg
- November 2025 (through 21 Nov) – 14 324 tablets dextromethorphan 15 mg; 3 612 tablets piracetam 1 200 mg

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These figures illustrate the growing off-label use of this protocol for difficult mood, anxiety, and trauma-spectrum presentations in everyday practice.

All patients provided written informed consent for publication of their clinical details. For those under 18, written consent was obtained from parents or legal guardians, and written assent was obtained from the adolescents themselves.

Results

Case 1: Treatment of Treatment-Resistent OCD Using the Fluoxetine + DXM Duo

An 18-year-old male with a long-standing diagnosis of obsessive–compulsive disorder (OCD) had been followed for several years in the public sector and was referred to our private clinic in March 2025 for persistent intrusive imagery and rumination despite trials of high-dose sertraline, aripiprazole augmentation, melatonin and methylphenidate. Initial assessment on 31 March 2025 documented marked affective comorbidity (PHQ-9 = 17; GAD-7 = 19) together with insomnia and violent recurrent mental images related to earlier school bullying. At that point his maintenance regimen from the public clinic comprised sertraline 150 mg/day, aripiprazole 15 mg/day, melatonin at bedtime and methylphenidate-LA 20 mg every morning.

Because intrusive thoughts dominated the clinical picture, fluoxetine 10 mg/day and dextromethorphan (DXM) 30 mg/day (15 mg twice daily) were introduced on top of his existing regimen as an evening combination, accompanied by low-dose lemborexant 2.5 mg and pregabalin 50 mg nightly for sleep. Within two weeks, on 15 April 2025, the patient reported substantially improved sleep continuity and a striking reduction in obsessive imagery; standardised ratings fell to PHQ-9 = 6 and GAD-7 = 4. The same doses of fluoxetine and DXM were therefore continued.

At review on 22 April 2025 the patient still described "repeated imagery, but milder." DXM was increased to 30 mg twice daily (15 mg × 2) while other agents were unchanged. By 20 May 2025 gastrointestinal discomfort from the generic fluoxetine was noted, yet mood remained stable (PHQ-9 = 8; GAD-7 = 7) and no suicidal intent was present. Because the obsessive images had again become bothersome, DXM was maintained at 60 mg/day and the fluoxetine formulation was changed rather than discontinued. Treatment adherence through late May and June resulted in continued, though incomplete, symptomatic relief: at the 17 June 2025 visit the patient described himself as "better, still a bit of imagery."

By 20 August 2025 depressive symptoms had resurfaced (PHQ-9 = 18; GAD-7 = 15) while sleep had again deteriorated. Fluoxetine was therefore titrated from 20 mg to 40 mg/day and sertraline was simultaneously reduced from 100 mg to 50 mg/day to streamline serotonergic therapy. Pregabalin 50 mg at bedtime was retained for nightmare reduction, and aripiprazole was increased by half a tablet to address the intrusive images. One month later, on 20 September 2025, the patient's affective scores improved (PHQ-9 = 12; GAD-7 = 7) and he described his obsessive imagery as "manageable."

Across months of observation, the patient's obsessive symptoms showed their most consistent attenuation during periods when fluoxetine and DXM were co-administered. Fluctuations in mood and anxiety correlated primarily with psychosocial stressors and adjustments to ancillary medications, whereas the severity of obsessive imagery appeared to track closely with the continuity and dosing of the fluoxetine + DXM combination. No negative neuropsychiatric effects linked to DXM were observed. These observations indicate that, in this patient with treatment-resistant OCD, the combination of fluoxetine and DXM resulted in a clinically significant reduction in obsessions that was not attained with high-dose selective serotonin reuptake inhibitor therapy and antipsychotic augmentation alone.

Case 2: Improving the Regimen by Adding Piracetam

In February 2025, a 31-year-old man with long-standing OCD came clinic looking exhausted. Two months earlier he had quit all of his medications from his previous psychiatrist on his own, hoping a break might help. Instead, his nights shattered into brief, restless stretches of sleep, and

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every time he closed his eyes he was ambushed by violent, self-harm images that played on an endless loop. When we ran the screening scales, his distress showed up clearly—PHQ-9 at 17 and GAD-7 at 21, both in the severe range.

Because he had previously responded only partially to selective-serotonin-re-uptake inhibitors (SSRIs), fluoxetine 20 mg/day was restarted together with low-dose risperidone 0.5 mg and dextromethorphan (DXM) 30 mg/day. DXM was titrated over the next three months to 120 mg/day (15 mg four times daily × 2). Concomitant hypnotics—lemborexant 1.25 mg, pregabalin 50 mg nocte and deanxit—were continued for insomnia and daytime anxiety.

Between March and June 2025 the combination of fluoxetine and DXM produced a clear attenuation of obsessive content: successive PHQ-9 scores fell to 7, and intrusive images became "less vivid and easier to dismiss." Nevertheless, periodic flares persisted, often triggered by alcohol use or occupational stress. On 17 June the patient again reported fragmented sleep and a return of suicidal imagery, prompting a brief increase in risperidone to 1 mg and the re-introduction of short-acting benzodiazepines. Although these spikes now resolved within days, he remained fatigued and complained of poor concentration at work.

In early September 2025, with fluoxetine 40 mg/day and DXM 120 mg/day firmly established, piracetam 600 mg twice daily (total 1.2 g/day) was added empirically for cognitive exhaustion and residual obsessiveness, while all other psychotropics were left unchanged. One week later, on 9 September, the patient reported noticeably lower mental fatigue, fewer ruminations and only occasional intrusive images; PHQ-9 fell from 8 to 6, and GAD-7 remained stable at 7. By 3 October he described himself as "much better with piracetam," able to stay at his desk even when exposed to usual contamination or symmetry triggers and, for the first time, able to let the images "pass like background noise." Objective scores confirmed the improvement (PHQ-9 = 5, GAD-7 = 7). He was working full days without interruptions and had resumed social meetings he had long avoided. On 30 October benefits were sustained. He volunteered that he had attempted—without success—to taper deanxit because he "felt no OCD at all" and feared rocking the boat. Ratings were unchanged (PHQ-9 = 5, GAD-7 = 7). Sleep remained consolidated on the existing hypnotic–pregabalin regimen, and no adverse effects such as headache, tremor or behavioural activation were noted.

Eight weeks after the piracetam add-on, the patient continued fluoxetine 40 mg/day, DXM 120 mg/day, piracetam 1.2 g/day, risperidone 2 mg/day, valproate 300 mg/day, pregabalin 50 mg nocte and deanxit one tablet daily. He reported only fleeting intrusive thoughts, no compulsive neutralising rituals and no suicidal ideation. Occupational functioning was described as "back to normal," and he was negotiating a graded reduction of sedative co-medication.

In this treatment-resistant case, augmentation of an SSRI–dextromethorphan regimen with piracetam was followed by an additional and durable reduction in obsessive symptoms, improved cognitive stamina and restoration of full occupational functioning. Fluoxetine plus high-frequency DXM provided a platform on which obsessive phenomena became shorter and less distressing but did not vanish. The subsequent introduction of low-dose piracetam coincided with a rapid, clinically significant fall in both subjective distress and screening scores that has endured for at least two months without dose escalation. No new adverse effects emerged after piracetam was started, nor were any other pharmacological or psychosocial variables altered during that window, supporting a specific therapeutic contribution from piracetam. This observation supports further exploration of piracetam as a potential adjunct for refractory OCD, particularly where intrusive imagery and mental fatigue remain problematic despite combined serotonergic and glutamatergic strategies.

Case 3: Treating OCD in an Adolescent with ASD with the Trio

A 16-year-old female secondary-school student with a documented history of autism spectrum disorder (ASD) was referred to our outpatient service in December 2024 for worsening obsessive–compulsive disorder (OCD). During the previous academic year she had become increasingly withdrawn, abandoning group projects, avoiding restaurants and repeatedly opening and closing her school locker until she felt "just right." Sleep-onset insomnia, contamination fears and intrusive

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doubts about personal adequacy dominated her evenings. Baseline rating scales confirmed moderate depressive and anxious symptomatology (PHQ-9 = 16, GAD-7 = 15).

She'd always shone at school—grade-8 piano, writing her own music, picking up new languages for fun. But her long-standing struggles with conversation, sensory overload and strict routines earned her an autism diagnosis when she was younger. Things got harder in Year 9 when a teacher she loved left without warning. She felt alone and her need for perfection went into overdrive. She was skipping classes a lot by April 2025, and she wouldn't sit down in public places because she was afraid of dirt. Counseling alone wasn't enough.

Short trials of low-dose risperidone and flupentixol–melitracen produced mild sedation but no meaningful change in ritualistic behaviour. In June 2025 we initiated a combined serotonergic–glutamatergic strategy: fluoxetine 10 mg daily together with dextromethorphan (DXM) 30 mg nightly. After six weeks the patient slept more soundly and irritability subsided, but hand-washing, device-wiping and locker-checking persisted. Symptom scores in August remained elevated (PHQ-9 = 18, GAD-7 = 18). Fluoxetine was therefore increased to 20 mg and DXM to 45 mg per day; risperidone was titrated to 1 mg nightly as antidopaminergic augmentation.

By early September the compulsions could be postponed for brief periods, yet the patient complained of mental fatigue, slowed homework completion and an "echo" of ruminative images that crowded out creative pursuits. Because cognitive lethargy can be particularly disabling for adolescents with ASD, we elected to introduce the nootropic agent piracetam as a potential pro-cognitive, anti-obsessional adjunct. The drug was started at 600 mg twice daily while the existing regimen of fluoxetine 20 mg, DXM 45 mg and risperidone 1 mg was maintained.

Four weeks later both patient and parent reported tangible improvement. She began opening classroom doors without immediate cleansing rituals, tolerated minor smudges on her mobile phone and completed group assignments with only minimal reassurance-seeking. Rating scales reflected the clinical impression (PHQ-9 = 7, GAD-7 = 9). Apart from two brief, self-limiting tremor episodes and menstrual irregularity, no adverse events emerged.At the six-week mark she returned to full school attendance, required no sick leave and re-engaged in piano practice and language study. Persistent gains allowed us to replace risperidone with aripiprazole 2.5 mg nightly to reduce metabolic liability. By mid-November the stabilised regimen consisted of fluoxetine 20 mg, DXM 45 mg, piracetam 1 200 mg and aripiprazole 2.5 mg daily; depressive symptoms remained in remission (PHQ-9 = 8) and anxiety was mild (GAD-7 = 7).The family declined further dose escalation, satisfied with the functional recovery achieved.

First of all, the stepwise combination of an SSRI (fluoxetine) and an NMDA-modulating antitussive (DXM) provided an initial reduction in hyperarousal but left cognitively taxing obsessions intact. The subsequent addition of piracetam coincided with sharper mental clarity, greater behavioural flexibility and a clinically meaningful fall in obsessive activity, suggesting a complementary mechanism of action. All in all, the full regimen was well tolerated in an adolescent with ASD, a population often sensitive to psychotropic side-effects. Minor tremor and menstrual irregularity were the only untoward effects observed.

For this teenager who lives with both OCD and autism, adding a small dose of piracetam to her fluoxetine-plus-dextromethorphan routine seemed to give her an extra boost. After the change she worried less, thought more clearly and got back to her usual activities. Her progress hints that piracetam could be a gentle, brain-friendly add-on for other neurodiverse adolescents who still struggle with stuck thoughts and mental tiredness even after standard medicines have helped.

Case 4: A Case Highlighting Serotonin-Toxicity Risk When Using the New Oral Regimen for OCD

Ms X, a 23-year-old woman with major depressive disorder and prominent obsessive–compulsive symptoms, came to clinic in early September 2025. She described years of fractured sleep, intrusive knife-related images and a growing tendency to avoid crowded places. Screening confirmed the clinical impression: her PHQ-9 measured 17 and her GAD-7 18, both in the moderate range.

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Because she had reacted poorly to paroxetine in the past from other doctors, we built a low-dose, multimodal plan that targeted mood and obsessions simultaneously. Each night she took fluoxetine 10 mg, two 15-mg tablets of dextromethorphan (DXM), risperidone 0.5 mg, piracetam 600 mg and lemborexant 2.5 mg; she also carried alprazolam 0.25 mg to use only during spikes of anxiety.

The first two weeks went well. Ms X began sleeping longer stretches, and her mood brightened enough that she ventured out with friends once or twice. Hoping to trim the remaining intrusive thoughts, we doubled the fluoxetine to 20 mg on 26 September and split DXM into two doses, raising the daily DXM total to 60 mg.

For three weeks she remained steady, until the evening of 17 October, when she sent an anxious text describing "frequent, very loud heart racing" and an inability to catch her breath that had persisted for several days. She reported no fever, tremor or sweating, but the tachycardia frightened her. The timing was suspicious: both fluoxetine—a selective serotonin-reuptake inhibitor and moderate CYP2D6 inhibitor—and DXM—a serotonin-reuptake inhibitor itself metabolised by CYP2D6—had been increased together. The picture fit a prodromal serotonin-toxicity state.

On 18 October we acted quickly. Fluoxetine 20 mg was stopped, and the original 10-mg dose reinstated; all other medications, including DXM, were left unchanged. Within 48 hours the tachycardia resolved completely. When Ms X returned on 24 October she was calm, euthymic and breathing comfortably. She reported better family harmony and had even begun teaching herself guitar. Her maintenance regimen now consisted of fluoxetine 10 mg, DXM 60 mg, risperidone 0.5 mg and piracetam 600 mg each night, along with lemborexant 2.5 mg for sleep and occasional alprazolam 0.25 mg. No further autonomic episodes have occurred.

This case shows how even a modest SSRI increase can, in the presence of DXM, edge a patient toward serotonin toxicity. The tight temporal link between the dose change and symptom onset, the solely autonomic presentation and the brisk recovery after lowering the SSRI all point to a pharmacological cause. Catching the reaction early spared Ms X the full serotonin-syndrome triad of mental-status change, neuromuscular hyperactivity and severe autonomic instability.

The take-home message is straightforward. DXM is more than cough medicine when paired with an SSRI: it blocks serotonin re-uptake, and fluoxetine slows its clearance, creating a potentially hazardous combination. Patients on both drugs should be urged to report new tachycardia, tremor or unexplained anxiety immediately, and clinicians should be ready to taper or stop one agent at the first hint of trouble.

Conclusion

The present case series illustrates the potential utility of a low-cost, orally administered glutamatergic protocol in treatment-resistant OCD. The pattern we saw resembled, on a smaller scale, the quick symptom relief reported with intravenous ketamine or add-on memantine, but relied on drugs that any community pharmacy can supply [5,6]. Pairing dextromethorphan (DXM) with the strong CYP2D6 inhibitor fluoxetine appeared to dial down intrusive thoughts and images within the first few weeks. That clinical course matches basic and clinical work showing that a brief block of NMDA receptors sparks a glutamate rebound and, through AMPA receptors, new synapse formation [8,9]. In two patients, adding a low dose of piracetam gave an extra lift—less cognitive "sticking," less mental exhaustion and better social ease—which fits with piracetam's gentle boost of AMPA current and BDNF signalling, all without notable side-effects at the doses we used.

That said, one individual did develop early serotonin-toxicity symptoms; DXM itself blocks serotonin re-uptake, and when its clearance is slowed by fluoxetine's CYP2D6 inhibition, the serotonergic load rises [7]. Rapid dose-reduction of the SSRI settled the reaction, underscoring the need for clear counselling and close follow-up.

The series has obvious limits: it is retrospective, uncontrolled and small; all patients were on other psychiatric medications; and formal OCD ratings such as the Y-BOCS were not collected at every visit. PHQ-9, GAD-7 scores and narrative notes therefore stand in for more granular data. Even so, the close temporal link between starting (or increasing) fluoxetine-boosted DXM and the drop in

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obsessive content, seen repeatedly across cases, supports a glutamatergic role that goes beyond the usual serotonergic or antipsychotic effects.

Taken together, these observations justify a more rigorous test of orally "boosted" DXM—with or without low-dose piracetam—as an accessible add-on for stubborn OCD. Randomised, controlled trials that include validated OCD scales, safety labs and perhaps magnetic-resonance spectroscopy of cortical glutamate could clarify efficacy, optimal dosing and long-term safety.

In four patients with treatment-resistant OCD, a combination of fluoxetine-potentiated dextromethorphan and, in selected cases, low-dose piracetam produced quick and lasting drops in intrusive obsessions, ruminations and day-to-day impairment. Benefits also touched anxiety, mood and, in one autistic patient, social engagement—without triggering mania or intolerable side-effects. One mild, self-limited episode of serotonin toxicity highlights the importance of careful titration and monitoring when mixing serotonergic and NMDA-modulating drugs. While clearly preliminary, the present series offers a proof of concept that targeting the NMDA–AMPA axis with readily available medicines may open a practical new lane in OCD treatment, meriting formal study.

Conflicts of Interest and Source of Funding Statement: None declared.

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