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Esketamine for The Treatment of Complex PTSD and Treatment-Resistant Depression: A Case Report

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ABSTRACT

Complex post-traumatic stress disorder (C-PTSD) frequently co-occurs with treatment-resistant depression (TRD), resulting in severe functional impairment, persistent suicidal ideation, and poor response to conventional treatments. We report the case of a 30-year-old woman with recurrent major depressive disorder, Complex PTSD, and borderline personality disorder, characterized by childhood neglect, sexual abuse, emotional dysregulation, recurrent self-harm, multiple psychiatric hospitalizations, and inadequate response to several pharmacological treatments. Because of persistent depressive symptoms and chronic suicidal ideation, intranasal esketamine was initiated at 56 mg for the first session and increased to 84 mg thereafter, alongside ongoing psychopharmacological treatment. Clinical outcomes were assessed using the Montgomery-Åsberg Depression Rating Scale (MADRS), Hamilton Anxiety Rating Scale (HAM-A), and Clinician-Administered PTSD Scale for DSM-5 (CAPS-5) over a 12-month follow-up. Sustained clinical improvement was observed, with MADRS decreasing from 36 to 4, HAM-A from 38 to 7, and CAPS-5 from 55 to 15, accompanied by complete remission of suicidal ideation. Improvements also involved core trauma-related symptoms, including intrusive recollections, hyperarousal, emotional dysregulation, and avoidance behaviors. This case suggests that intranasal esketamine may represent a promising therapeutic option for patients with treatment-resistant depression and comorbid Complex PTSD, producing sustained benefits across depressive, anxiety, and trauma-related symptom domains. Although limited by the single-case design, these findings support further investigation of esketamine in complex trauma-related psychopathology.

Keywords: *Esketamine, Treatment-Resistant Depression, Complex PTSD, Borderline Personality Disorder, Suicidal Ideation, Trauma*

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Introduction

Complex Post-Traumatic Stress Disorder (C-PTSD) represents a severe and chronic trauma-related condition characterized not only by the core symptoms of Post-Traumatic Stress Disorder (PTSD)—including intrusive memories, avoidance, hyperarousal, and negative alterations in cognition and mood—but also by pervasive disturbances in self-organization, emotional dysregulation, negative self-concept, and interpersonal dysfunction. Individuals with C-PTSD frequently experience substantial functional impairment, chronic psychological distress, elevated rates of psychiatric comorbidity, and poorer treatment outcomes compared with patients affected by PTSD alone (American Psychiatric Association, 2013; Rothärmel M *et al*., 2022; Rothärmel M *et al*., 2026).

Among psychiatric comorbidities, Major Depressive Disorder (MDD) is particularly common. Epidemiological studies indicate that approximately half of patients with PTSD also meet criteria for major depressive disorder, and this coexistence is associated with greater illness severity, increased functional impairment, higher rates of treatment resistance, and a markedly elevated risk of suicidal behavior. (Bedard-Gilligan *et al*., 2015; Raab *et al*., 2015; Rytwinski *et al*., 2013). Trauma exposure itself has been recognized as a significant risk factor for treatment-resistant depression (TRD), suggesting that trauma-related mechanisms may contribute to the persistence of depressive symptoms even when conventional antidepressant treatments are administered (Rothärmel *et al*., 2026).

Current therapeutic strategies for PTSD and C-PTSD primarily rely on trauma-focused psychotherapies, including prolonged exposure, cognitive processing therapy, and Eye Movement Desensitization and Reprocessing (EMDR). Although these interventions demonstrate robust efficacy, a substantial proportion of patients either fail to respond adequately, discontinue treatment prematurely, or continue to experience clinically significant residual symptoms. Pharmacological treatments, mainly based on antidepressants, generally produce modest benefits and often fail to address the complexity of trauma-related psychopathology, particularly in patients presenting with severe emotional dysregulation, dissociative symptoms, personality pathology, or treatment-resistant depression (Rothärmel *et al*., 2026).

Esketamine, the S-enantiomer of ketamine and a non-competitive N-methyl-D-aspartate (NMDA) receptor antagonist, has emerged as a rapid-acting treatment for TRD. Through modulation of glutamatergic neurotransmission, increased AMPA receptor activation, enhancement of brain-derived neurotrophic factor (BDNF) signaling, and promotion of synaptogenesis within cortico-limbic circuits, esketamine appears capable of inducing rapid antidepressant and anti-suicidal effects while

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simultaneously promoting neuroplasticity (Duman *et al*., 2016). These mechanisms have generated growing interest regarding its potential utility beyond depression, particularly in trauma-related disorders.

Recent evidence suggests that ketamine and esketamine may exert beneficial effects on PTSD symptomatology. Randomized controlled trials of intravenous ketamine have demonstrated rapid reductions in intrusive symptoms, avoidance behaviors, and hyperarousal in patients with chronic PTSD. (Feder *et al*., 2014; Feder *et al*., 2021). Moreover, emerging observational studies involving intranasal esketamine in patients with comorbid TRD and PTSD have reported clinically meaningful improvements in both depressive and trauma-related symptoms (Rothärmel *et al*., 2022). Interestingly, some patients experience trauma re-experiencing phenomena or flashbacks during esketamine sessions, which may reflect activation and reconsolidation of traumatic memory networks rather than simple adverse effects (Oquendo *et al*., 2016). When appropriately managed within a supportive therapeutic setting, these experiences have been hypothesized to contribute to symptom improvement through mechanisms related to emotional processing and memory reconsolidation (Feder *et al*., 2021).

An additional aspect of particular relevance concerns the frequent coexistence of trauma-related disorders with Borderline Personality Disorder (BPD). Emotional dysregulation, impulsivity, self-harm behaviors, and chronic suicidality represent shared clinical dimensions that substantially complicate treatment.

Recent naturalistic evidence suggests that intranasal esketamine may be associated with significant reductions in suicidal ideation, suicidal behaviors, and deliberate self-harm even among patients with comorbid BPD, supporting the hypothesis that glutamatergic modulation may positively influence affective regulation and behavioral control in highly complex clinical populations (Raffone *et al*., 2026a).

Against this background, we report the case of a young woman affected by treatment-resistant depression, Complex PTSD, and borderline personality disorder who experienced a marked and sustained clinical improvement following treatment with intranasal esketamine. This case contributes to the emerging literature exploring the role of esketamine in trauma-related psychopathology and highlights its potential therapeutic value in patients characterized by severe psychiatric comorbidity, chronic suicidality, and limited response to conventional pharmacological interventions.

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Case Presentation: A 30-year-old woman presented with a history of three previous major depressive episodes and longstanding symptoms consistent with C-PTSD and BPD. The diagnosis of Complex PTSD was established according to ICD-11 diagnostic criteria and based on longitudinal clinical assessment performed by experienced psychiatrists (World Health Organization, 2019). Psychopathology began at age 18, manifesting as emotional dysregulation, self-harming behavior, and affective instability. The patient had undergone multiple psychiatric hospitalizations and a two-year residential treatment program. Trauma history included childhood neglect and maltreatment, domestic violence, and sexual abuse by a family acquaintance at age 16. Her family history was positive for mood disorders on the maternal side and substance use disorders on the paternal side.

At presentation in 2023, the patient exhibited a chronic and severe depressive episode beginning in mid-2022, characterized by persistent suicidal ideation, psychomotor slowing, anhedonia, apathy, sleep disturbances, emotional lability, flashbacks, and symptoms of generalized anxiety. The SCID-II interview confirmed comorbid borderline personality disorder. The clinical picture met diagnostic criteria for recurrent Major Depressive Disorder and Complex PTSD according to ICD-11 (World Health Organization, 2019), and Borderline Personality Disorder according to DSM-5 (American Psychiatric Association, 2013).

Following insufficient response to multiple pharmacological regimens including SSRIs (citalopram, sertraline), trazodone, benzodiazepines (lorazepam, delorazepam), and mood stabilizers (gabapentin, valproate), the patient was referred to the Esketamine Clinic in Pavia. Esketamine nasal spray was initiated at a dose of 56 mg biweekly, increased to 84 mg from the second administration. Concomitant medications included citalopram 40 mg/day, aripiprazole LAI 400 mg monthly, and clonazepam oral drops (10 drops TID) (Rothärmel *et al*., 2022).

Methods: The patient was prospectively monitored over a 12-month follow-up period to evaluate both the short- and long-term clinical effects of intranasal esketamine treatment. Symptom severity was assessed at baseline (T0), after 1 week (T1), 1 month (T2), 3 months (T3), 6 months (T4), and 12 months (T5) using standardized clinician-administered rating scales. Depressive symptoms were evaluated with the Montgomery–Åsberg Depression Rating Scale (MADRS) (Montgomery *et al*., 1979), anxiety symptoms with the Hamilton Anxiety Rating Scale (HAM-A) (Hamilton *et al*., 1959), and post-traumatic symptomatology with the Clinician-Administered PTSD Scale for DSM-5 (CAPS-5) (Weathers *et al*., 2018). The diagnosis of Borderline Personality Disorder was confirmed through the Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II) (First, 1997). This longitudinal assessment framework allowed for the systematic evaluation of symptom trajectories across depressive, anxiety, and trauma-related domains throughout both the induction and maintenance phases of treatment.

All study procedures adhered to the principles of Good Clinical Practice (GCP) and the principles of the Declaration of Helsinki. Ethical approval was obtained from the Pavia Ethics Committee (Opinion No. 84157/21, 27 August 2021; amendment No. 0102231/21). Written informed consent for publication of anonymized clinical information was obtained from the patient.

Results: Marked clinical improvement was observed during both the induction and maintenance phases of esketamine therapy. Symptom reduction was tracked using validated rating scales:

- MADRS total score decreased from 36 at baseline (T0) to 4 at one year (T5)

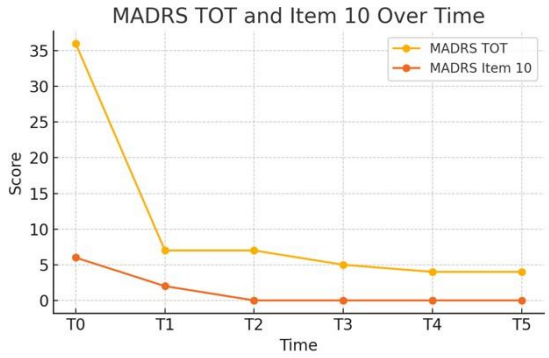

Figure 1: MADRS Total and Item 10 Over Time.

- Hamilton Anxiety Rating Scale (HAM-A) decreased from 38 to 7

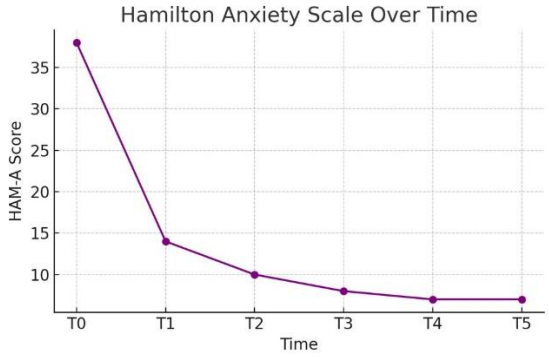

Figure 2: Hamilton Anxiety Scale Scores Over Time.

- CAPS-5 for PTSD symptomatology decreased from 55 (moderate PTSD) to 15 (few symptoms)

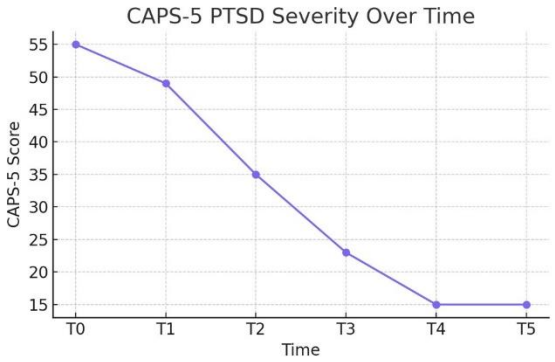

Figure 3: CAPS-5 PTSD Symptom Severity Over Time.

Table 1: CAPS-5: PTSD severity according to total score (adapted from Weathers *et al.*, 2018).

Time Point	CAPS-5 Total Score	PTSD Severity
T0	55	Moderate PTSD
T1	49	Moderate PTSD
T2	35	Mild PTSD
T3	23	Mild PTSD
T4	15	Asymptomatic / Few symptoms
T5	15	Asymptomatic / Few symptoms

- 0-19: Asymptomatic/Few symptoms
- 20-39: Mild / Subthreshold PTSD symptoms
- 40-59: Moderate PTSD symptoms
- 60-79: Severe PTSD
- ≥80: Extreme PTSD symptoms

Figures 1–3 demonstrate the progressive improvements observed across all dimensions.

Discussion

This case report describes the successful use of intranasal esketamine in a patient with treatment-resistant depression (TRD), Complex Post-Traumatic Stress Disorder (C-PTSD), and borderline personality disorder (BPD), resulting in a marked and sustained improvement in depressive, anxiety, and trauma-related symptoms over a 12-month follow-up period.

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The coexistence of PTSD and major depressive disorder represents a particularly challenging clinical condition (Rytwinski *et al*., 2013; Bedard-Gilligan *et al*., 2015; Raab *et al*., 2015; Oquendo *et al*., 2005).

In the present case, conventional pharmacological strategies, including multiple antidepressants, benzodiazepines, and mood stabilizers, failed to provide meaningful therapeutic benefit. Treatment with intranasal esketamine was followed by a progressive reduction in depressive symptom severity, accompanied by marked improvements in anxiety, emotional dysregulation, trauma-related psychopathology, and suicidal ideation. Importantly, the marked reduction in CAPS-5 scores suggests that the therapeutic effects extended beyond depressive symptomatology to encompass core PTSD dimensions, including intrusive recollections, hyperarousal, and avoidance.

The mechanisms underlying these improvements remain speculative but are supported by an emerging body of literature. Esketamine exerts its antidepressant effects primarily through non-competitive antagonism of NMDA receptors, resulting in increased glutamatergic transmission, AMPA receptor activation, enhanced brain-derived neurotrophic factor (BDNF) signaling, and increased synaptogenesis within cortico-limbic circuits involved in emotional regulation. These neuroplastic changes are believed to contribute to the rapid reduction of depressive symptoms and suicidal ideation observed in treatment-resistant populations (Duman *et al*., 2016).

Beyond its antidepressant effects, growing evidence suggests that ketamine and esketamine may influence trauma-related psychopathology. Randomized controlled trials of intravenous ketamine in chronic PTSD have demonstrated rapid reductions in PTSD symptom severity, (Feder *et al*., 2014; Feder *et al*., 2021) including intrusive recollections, avoidance, and hyperarousal symptoms. Feder and colleagues (Feder *et al*., 2021) reported significant improvements following both single and repeated ketamine infusions, supporting the hypothesis that glutamatergic modulation may directly affect neural circuits implicated in traumatic memory processing. Similar findings were observed in military populations with chronic PTSD, where ketamine produced clinically meaningful symptom reductions compared with placebo.

Recent theoretical models have proposed that esketamine may induce a temporary "window of neuroplasticity," (Raffone *et al*., 2026b). facilitating the modification of maladaptive emotional and autobiographical memory networks. Although direct evidence remains limited, this hypothesis is particularly relevant in complex trauma-related disorders. Trauma memories are often characterized by persistent emotional salience, fragmented processing, and impaired contextual integration. Through

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enhancement of synaptic plasticity and modulation of fear-related neural circuitry, esketamine may facilitate adaptive reprocessing of traumatic experiences and reduce the affective intensity associated with traumatic recollections. Current evidence supports mechanistic plausibility for this model, although definitive confirmation is still lacking.

The marked improvement observed in this patient is also consistent with emerging clinical reports describing beneficial effects of esketamine in patients with comorbid PTSD and TRD. Rothärmel and colleagues reported a multicenter series of patients with PTSD and treatment-resistant depression receiving intranasal esketamine, demonstrating clinically significant improvements in both depressive and trauma-related symptoms (Rothärmel *et al*., 2022). Interestingly, many patients experienced transient trauma-related flashbacks during treatment sessions, phenomena that often diminished over time and were not necessarily associated with poorer outcomes. The authors hypothesized that reactivation of traumatic memories during esketamine treatment may reflect engagement of memory reconsolidation mechanisms and could potentially facilitate therapeutic processing when appropriately managed.

Another relevant aspect of the present case concerns the presence of borderline personality disorder. Emotional dysregulation, impulsivity, unstable interpersonal relationships, and recurrent self-harm behaviors frequently complicate the treatment of both PTSD and depression (Raffone *et al*., 2026a).

Recent research has further highlighted the importance of psychological dimensions such as mentalization capacity, cognitive rigidity, psychache, and suicidality in shaping clinical outcomes during esketamine treatment, supporting increasingly personalized approaches to treatment-resistant mood disorders (Olivola *et al*., 2026).

Historically, patients with significant personality pathology have often been excluded from clinical trials evaluating novel antidepressant treatments. Nevertheless, preliminary evidence suggests that esketamine may improve affective regulation and reduce emotional reactivity through modulation of cortico-limbic networks implicated in impulsivity and emotional control. The substantial reduction in affective instability and suicidal ideation observed in this patient supports this hypothesis and suggests that borderline personality pathology should not necessarily be considered a barrier to treatment (Duman *et al*., 2016; Duek *et al*., 2023).

This observation is consistent with emerging evidence suggesting that psychological factors traditionally associated with poor outcomes, including elevated psychache, impaired mentalization, cognitive rigidity, and chronic suicidality, do not necessarily preclude a favorable response to esketamine

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treatment and may instead represent clinically relevant domains for treatment personalization (Olivola *et al*., 2026).

In our opinion, the most clinically meaningful finding is not merely the magnitude of symptom reduction, but its persistence over time. In patients with complex trauma, therapeutic success is often limited by recurrent emotional dysregulation and rapid clinical relapse. The sustained stability observed throughout one year of follow-up raises the intriguing possibility that maintenance esketamine may support a more enduring reorganization of emotional functioning, extending its effects beyond antidepressant efficacy alone. Although this interpretation remains speculative, it aligns with the emerging view that glutamatergic modulation may facilitate adaptive neuroplastic processes capable of supporting long-term recovery in carefully selected patients.

Several limitations should be acknowledged. As a single case report, causal inferences cannot be established and spontaneous improvement cannot be completely excluded. Furthermore, concomitant pharmacological treatment may have contributed to the observed outcomes. The absence of structured measures of dissociation, emotional regulation, and psychosocial functioning limits the ability to fully characterize the mechanisms underlying clinical improvement. Nevertheless, the longitudinal design, standardized psychometric assessments, and extended follow-up strengthen the clinical relevance of the observations.

Overall, this case contributes to the growing literature suggesting that intranasal esketamine may represent a valuable therapeutic option for patients with treatment-resistant depression and comorbid trauma-related disorders. Future prospective studies involving larger samples, standardized PTSD assessments, and neurobiological outcome measures are needed to clarify the specific role of esketamine in the treatment of Complex PTSD and to identify predictors of response in patients with highly complex clinical presentations.

This case report describes substantial and sustained clinical improvement following intranasal esketamine treatment in a patient with comorbid Complex Post-Traumatic Stress Disorder (C-PTSD), treatment-resistant depression (TRD), and borderline personality disorder (BPD). Treatment was associated with marked reductions in depressive symptoms, anxiety, trauma-related psychopathology, and suicidal ideation, with benefits maintained throughout a 12-month follow-up. Importantly, the improvement extended beyond mood symptoms to include core PTSD dimensions, such as intrusive recollections, hyperarousal, emotional dysregulation, and avoidance behaviors.

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These findings add to the growing evidence that esketamine may exert beneficial effects not only on treatment-resistant depression but also on trauma-related psychopathology, potentially through glutamatergic modulation, enhanced neuroplasticity, and facilitation of adaptive emotional processing. Such mechanisms may be particularly relevant in patients with complex trauma histories, severe affective dysregulation, and personality pathology, who frequently show limited response to conventional treatments.

Although limited by the single-case design, the magnitude and durability of the clinical response highlight the potential value of esketamine in highly complex and treatment-refractory presentations. The favorable outcome observed despite comorbid borderline personality disorder further suggests that personality pathology should not automatically preclude consideration of esketamine treatment (Rothärmel *et al*., 2022; Raffone *et al*., 2026). Future prospective studies involving larger cohorts are warranted to clarify long-term efficacy, safety, and predictors of response, including clinical and psychological factors, and to explore the integration of esketamine with trauma-focused psychotherapeutic interventions as part of personalized treatment strategies (Olivola *et al*., 2026; Raffone *et al*., 2026).

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