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Short Communication

Role of Ketamine in Psychopharmacotherapy for Eating Disorders: A Brief Note

Madeline Bratyle*

Department of Pharmacotherapy, University of Utah College of Pharmacy, Salt Lake City, USA

DESCRIPTION

Eating Disorders (EDs) are highly prevalent, disabling and potentially fatal psychiatric illnesses characterized by abnormal eating and weight disturbances. They are etiologically complex and multifactorial in nature, often leading to severe psychological and somatic complications, marked functional impairment, and poor quality of life and overall prognosis. Approximately 8%-15% of children and adolescents and 16% of adults are affected by EDs, with weighted population means of lifetime prevalence at 1.6% for anorexia nervosa. However, the prevalence of EDs is greatly underestimated, primarily due to variable diagnostic classifications, underreporting, and lack of research funding. Moreover, EDs are associated with significantly elevated morbidity and mortality, compared with the general population, with the highest rates occurring in AN. Anorexia nervosa, in particular, carries a 12-fold increased risk of death-higher than any other psychological condition-to which low body mass index, poor social adjustment, and alcohol dependence have been reported as significant predictors. Mortality rates for EDs are further complicated by concomitant psychiatric comorbidities (e.g., anxiety, depression, and substance abuse), as well as symptom persistence. While various models have attempted to explain ED pathogenesis, the mechanisms subserving disease onset, progression, and maintenance remain not fully understood. Notwithstanding, several hypothesis surround the neurobiology of EDs, which is supported by a growing body of literature. Currently, the primary care pathway for EDs is psychological and dietetic intervention, followed by psychotropic medication. Treatment is generally provided on an outpatient basis, with medically compromised individuals recommended to higher levels of ED care, including intensive outpatient, partial hospitalization, and residential programs [1,2].

Use of ketamine in ED

Few studies have examined the therapeutic use of ketamine for EDs, which are limited to case series and reports, and are focused on AN over other primary (BN and BED) and secondary (pica, RD, and ARFID) subgroups. Patients received 2–15 ketamine infusions scheduled at 5-21-day intervals, dependent upon clinical response, and were delivered at 20 mg/h over 10 h. This was an intense drug regimen-relative to current studies on ketamine and mental health previously used to treat postoperative pain and acute war injuries. Marked and sustained remissions were observed in responders (n=9) compared to non-responders (n=6), with no-to-minimal disease activity at 7-24 months follow-up. Moreover, responders showed significant reductions in obsessive compulsive neurosis ($p<0.001$), in addition to increased weight acceptance, partial-to-complete weight restoration, and resolved amenorrhea. No significant improvements were reported for non-responders. Investigators attributed this result to premature relapses following treatment, during which compulsive drives may have been re-established, and/or the result of insufficient doses of nalmefene. Overall, clinical response (≥ 50% reduction in symptom severity) was associated with AN subtype. A recent longitudinal case series similarly produced positive outcomes, showing repeat dosing of ketamine to be moderately effective in four patients diagnosed with severe and enduring AN-R or EDNOS-BP and comorbid TRD of 11.0 years ± 1.4. Patients had previously completed partial hospitalization programs for their ED, reported persistent negative affectivity, and failed several trials of mono-therapy antidepressants of adequate dose and duration. Ketamine was administered intramuscularly and/or intravenously at 0.5 mg/kg over 30–90 min, with subsequent doses titrated to 0.8–0.9 mg/kg depending on treatment toleration and response. Repeat dosing was scheduled at 4–6-week intervals spanning 12+ months, resulting in clinically meaningful changes in depression, as well as modest changes in anxiety and disordered eating. Interestingly, patients with AN-R demonstrated robust and sustained responses, compared to their EDNOS-BP counterparts, in addition to marked improvements in psychosocial functioning and quality of life trajectories. The differential degree of ketamine efficacy between ED subgroups merits further investigation [2-4].

Future contexts and directions

Future studies should be aimed at investigating, setting, and optimizing the ketamine dose, duration, and frequency of ED to support clinical recommendations and evidence-based practice

Correspondence to: Dr. Madeline Bratyle, Department of Pharmacotherapy, University of Utah College of Pharmacy, Salt Lake City, USA, E-mail: munaalwaeli338@gmail.com

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guidelines. Therefore, open pilot studies and statistically supported feasibility, randomization and conduct studies are justified. In addition, empirically derived standardized treatment regimens and result measurements have been proposed to facilitate comparisons between studies. Longitudinal assessment is further recommended to characterize the clinical response of patients with severe and persistent illness. Given that ketamine can (1) normalize ED-related glutamatergic dysfunction, (2) promote synaptogenesis and neuroplasticity, in important post-dose windows, (3) Short time to recurrence (2-6 weeks), it is most important to combine ketamine with psychotherapy, either supplementally or in combination. The authors hypothesized that ketamine and psychotherapy act synergistically, treatment enhances the response to treatment, and repeated sessions are responsible for the persistence of the effect. In addition, the "appearance" of ketamine, characterized by euphoria, lucid dreaming, and hallucinations, can promote therapeutic relationships, patient-care provider ties, and ultimately behavioural changes. Although course of treatment is designed to allow flexible dosing and personalization, KAP usually consists of preparatory psychotherapy (step 1), ketamine dosing (step 2), and integrated psychotherapy (step 3). Follow the 3-step model. Discussing the patient's motives, intentions, and expectations for treatment, assessing their current psychophysiological status, and providing a therapeutic environment that facilitates the ketamine experience (i.e., "settings and settings") should be considered prior to treatment. It's an important factor to do. It is also advisable to deal with various psychotherapeutic approaches in this context. Over the years, it has become increasingly diverse in cognitive-behavioural, humanity, functional analysis, and body-based interaction therapies.

CONCLUSION

The evidence presented here provides a conceptual but concise summary of the use of ketamine in the treatment of ED. Although the relevant literature is still scarce, studies show the potential for treatment of this complex and almost unexplored population. In particular, ketamine provides maximum benefit to clinical non-responders who are resistant to the psychological, dietary, and pharmacological interventions used in standard practice and are prone to develop long-term ED pathology, Overall subgroups (significant in BN, BED, and AFRID) and diagnosis-dependent severity (mild, moderate, severe, and extreme) and lifetime (children to elderly). This data can then be used to create safety profiles, optimize dosing, and inform targeted treatment strategies at the individual patient level. Adjuvant and combination therapies, especially KAP, also provide empirical research and the opportunity to determine which situations and intervention settings are most appropriate for ketamine.

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