

CASE REPORT

Biochemical Stabilization Preceding Psychological Engagement in Alcohol Use Disorder: A Case Report

Author(s): Heather Dale, MS, MPA, NTP, CARNC-II¹

1. Independent Clinical Practice, Addiction Recovery Nutrition and Biochemical Support, Denver, Colorado, USA; Correspondence: heather@heatherdalecoaching.com

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ABSTRACT

Background: Alcohol use disorder (AUD) is frequently characterized by repeated relapse despite psychological insight, motivation, and engagement in psychotherapy. Chronic alcohol exposure is associated with metabolic instability, micronutrient depletion, and impaired stress tolerance, which may limit the real-time effectiveness of psychological interventions during high-risk periods.

Case Report: Here, I report on the case of a woman in her early 50s with a 25-year history of AUD marked by recurrent cycles of abstinence and relapse. Despite significant insight and a prior three-year period of sobriety, she experienced predictable relapse vulnerability in the early evening, characterized by anxiety and physiological dysregulation rather than hedonic craving. A targeted orthomolecular stabilization approach was implemented over a 16-month period, emphasizing structured protein-forward meals alongside symptom-guided amino acid, micronutrient, and omega-3 essential fatty acid support. Craving intensity markedly decreased within 10 days of intervention. Sleep continuity improved, anxiety-driven urges became manageable using non-alcohol strategies, and engagement in psychotherapy increased. At 16 months of follow-up, the patient remained abstinent.

Conclusion: This case illustrates how restoration of biological capacity through nutritional and amino acid support may precede and enable sustained psychological engagement and relapse prevention in alcohol use disorder.

Keywords: Alcohol Use Disorder; Amino Acids; Blood Sugar Regulation; Orthomolecular Medicine; Nutritional Psychiatry; Relapse Prevention

INTRODUCTION

Alcohol use disorder (AUD) remains associated with high relapse rates, even among individuals who demonstrate insight, motivation, and engagement in psychotherapy (Volkow et al., 2016). While psychological and behavioral interventions remain foundational, their effectiveness may depend partly on whether the neurobiological capacity required to tolerate stress, regulate emotion, and implement coping strategies is physiologically available during high-risk periods.

Chronic alcohol use is known to disrupt blood glucose regulation, deplete key micronutrients (including B vitamins and magnesium), and alter amino acid availability relevant to neurotransmitter synthesis (Martin et al., 2003; Thomson & Marshall, 2006; White & Sirohi, 2024). Clinically, these disruptions may manifest as fatigue, anxiety, impaired stress tolerance, emotional volatility, sleep disruption, and heightened relapse vulnerability. Many patients with AUD describe relapse not as a conscious decision to pursue pleasure, but as an urgent attempt to relieve internal tension, dysregulation, exhaustion, or anxiety arising during predictable physiological states (Koob & Le Moal, 2001; Oba-Yamamoto et al., 2021). This case report proposes that biochemical stabilization – through targeted nutritional and amino acid

support – may be a necessary precondition for effective psychological engagement in alcohol use disorder.

Orthomolecular approaches aim to restore depleted biochemical substrates using nutrients normally present in the human body (Pfeiffer, 1975). While mechanistic and deficiency-based literature is substantial, there remains a relative scarcity of detailed clinical case reports documenting how targeted biochemical stabilization may alter relapse vulnerability and engagement with psychological treatment.

PATIENT INFORMATION

The patient is a perimenopausal woman in her early 50s with a more than 25-year history of AUD. Her course was characterized by repeated cycles of abstinence followed by relapse. Periods of abstinence typically lasted weeks to months, with one extended period of approximately three years more than 15 years prior to presentation. Since that time, she described a persistent “merry-go-round” of relapse and recovery, which she found increasingly distressing and demoralizing. In the years preceding presentation, the patient also reported increasing sleep disruption, heightened stress sensitivity, and worsening anxiety during the perimenopausal transition. These symptoms appeared to compound existing relapse vulnerability, particularly during periods of fatigue and emotional overwhelm.

Relapse episodes were unpredictable in duration. Once alcohol use resumed, she could not reliably anticipate whether drinking would be limited to a single evening or escalate into weeks or months of daily use before regaining control.

She was married and parenting one child at home. She had previously engaged in psychotherapy and recovery-oriented supports and demonstrated high insight and motivation to stop drinking. Upon clinical presentation, she expressed desperation to interrupt the repeated relapse cycle.

CLINICAL FINDINGS

A consistent relapse pattern emerged over time. The patient reported that her highest-risk window occurred in the early evening, typically after returning home from work. During this period, she often experienced a combination of physical exhaustion, psychological stress, and heightened anxiety. These states were accompanied by strong urges

to drink, experienced not primarily as reward-seeking, but as relief-seeking responses to internal tension, anxiety, exhaustion, and physiological dysregulation.

Additional features included:

- Irregular eating patterns earlier in the day
- Tendency to skip or under-eat breakfast
- Anxiety-driven cravings rather than reward-seeking
- Fragmented sleep with frequent nighttime awakenings, particularly during periods of drinking
- Increasing difficulty tolerating stress despite psychological insight

Baseline (Pre-intervention):

- 25+ years of AUD with repeated abstinence-relapse cycles
- Longest prior sobriety: 3 years, more than 15 years ago
- Frequent early-evening cravings associated with stress and fatigue
- Anxiety-driven urges and poor sleep continuity

Intervention Period (Weeks 1–2):

- Immediate implementation of structured meals with non-negotiable protein intake
- Introduction of targeted amino acid and micronutrient support
- Rapid reduction in craving intensity

Early Follow-Up (First Month):

- One brief drinking episode within the first month
- Post-relapse review and refinement of “crisis protocols”
- Temporary increase in anxiolytic amino acid support

Ongoing Follow-Up (Months 2–16):

- No further alcohol use
- Sustained sleep improvement
- Increased engagement with therapy and social activities
- Continued abstinence at 16 months

Diagnostic Assessment

Formal laboratory testing was not central to the clinical assessment. Instead, a capacity-based framework was applied, informed by symptom patterns and alcohol use history.

Clinical considerations included:

- **Blood sugar instability**, suggested by late-day fatigue, anxiety, and urgency that improved rapidly with regular protein intake (Lieber, 2003; Oba-Yamamoto et al., 2021).
- **Neurotransmitter dysregulation**, particularly affecting serotonergic and inhibitory tone, consistent with anxiety-driven cravings. Chronic alcohol exposure is known to alter reward, stress, and inhibitory systems, including dopaminergic and gamma-aminobutyric acid-ergic pathways (Koob & Le Moal, 2001; Volkow et al., 2016).
- **Micronutrient depletion**, commonly associated with long-term alcohol use and stress, particularly involving B vitamins and magnesium, which are essential for neurotransmitter synthesis and nervous system regulation (Martin et al., 2003; Thomson & Marshall, 2006; White & Sirohi, 2024).

Rather than framing relapse as a failure of motivation or insight, the working hypothesis was that repeated episodes occurred during predictable physiological states in which stress tolerance and inhibitory control were compromised (Koob & Le Moal, 2001; Volkow et al., 2016).

Therapeutic Intervention

A foundational intervention focused on stabilizing blood sugar through consistent protein intake:

- **Non-negotiable breakfast** containing protein
- **Protein every 4 hours**, with a minimum target of 20 grams per meal
- Emphasis on regular meals to prevent late-day metabolic crashes

This structure was framed as recovery infrastructure rather than dietary optimization.

Amino Acid Support

Amino acids were selected based on the patient's symptom profile and used in a symptom-guided manner to support mood regulation, stress tolerance, and craving management.

Interventions included:

- **5-hydroxytryptophan** (5-HTP; 100 mg nightly) to support serotonergic tone, sleep continuity, and mood stability (Birdsall, 1998)
- **L-theanine** (200 mg twice daily) for anxiety modulation, used both regularly and during acute craving states (Hidese et al., 2019)
- **L-glutamine** (2–5 g as needed) during craving episodes, including in beverage form (Rogers & Pelton, 1957; Williams, 1967)

During periods of increased vulnerability, L-theanine dosing was temporarily increased to 200 mg three times daily. Interventions were adjusted based on response and tolerability.

Micronutrients and Essential Fats

Additional support included:

- A comprehensive daily multivitamin/mineral
- **B-complex supplementation**, recognizing the established role of B vitamins in neurological function and the high prevalence of deficiency in individuals with AUD; dosed as a high-potency broad-spectrum B-complex (approximately 25–50 mg of most B vitamins) (Martin et al., 2003; Thomson & Marshall, 2006)
- **Omega-3 essential fatty acids**, to support neuroinflammation modulation and neuronal membrane function, dosed at approximately 2000 mg combined eicosapentaenoic acid/docosahexaenoic acid daily (Su et al., 2015)
- **Magnesium glycinate** (400 mg nightly), to support nervous system regulation and sleep quality (Rawji et al., 2024), given the high prevalence of magnesium depletion in individuals with AUD (Rivlin, 1994)

All interventions were individualized and adjusted based on response and tolerability. The patient was seen weekly during early stabilization, then monthly through the 16-month follow-up period.

Follow-Up and Outcomes

The patient reported a rapid reduction in cravings following stabilization of meal timing and protein intake. Within approximately 10 days, cravings that previously felt overwhelming became manageable and responsive to non-alcohol interventions such as amino acids.

Sleep improved substantially. The patient reported frequent middle-of-the-night awakenings, heightened nighttime anxiety, and worsening sleep continuity during the perimenopausal transition before later transitioning to sleeping through the night more consistently as stabilization progressed.

Engagement in psychotherapy improved markedly. The patient described an increased ability to tolerate emotionally challenging sessions, apply therapeutic tools during stress, and remain regulated between sessions. As stability increased, she also re-engaged socially, joining clubs and reviving personal interests that had previously fallen away during cycles of drinking.

After a single drinking episode within the first month of care, no further alcohol use occurred. At 16 months of follow-up,

the patient remained abstinent and reported sustained improvements in stress tolerance, sleep, and quality of life.

DISCUSSION

This case highlights a clinically common but under-documented phenomenon: repeated relapse in AUD despite insight, motivation, and psychological support. In this patient, relapse vulnerability clustered around predictable physiological states – particularly early evening periods of fatigue, anxiety, and metabolic depletion.

Importantly, cravings were experienced not primarily as reward-seeking, but as relief-seeking responses to internal tension, anxiety, exhaustion, and physiological dysregulation. This distinction may reflect a shift from hedonic to regulatory drivers of alcohol use, in which alcohol functions as a rapid means of restoring perceived internal equilibrium rather than producing pleasure.

Biological Capacity as a Precondition for Psychological Work

The intervention did not replace psychotherapy; rather, it appeared to restore the patient's capacity to benefit from it. Chronic alcohol use is associated with dysregulation in stress and reward systems, which can impair executive function and emotional regulation during high-risk periods (Koob & Le Moal, 2001; Volkow et al., 2016).

Once blood sugar stability and anxiety regulation improved, psychological strategies became accessible during moments when they had previously failed. Clinically, this pattern is frequently observed: patients may possess insight and motivation, yet remain unable to implement behavioral strategies during periods of physiological distress.

This capacity-based framing offers a complementary perspective to existing psychological models of relapse, emphasizing the role of an individual's physiological state in determining access to coping strategies and behavioral control.

Blood Sugar Stability and Craving Vulnerability

The rapid reduction in cravings following structured protein intake suggests that metabolic instability may play a significant role in relapse risk for some individuals with AUD. Fluctuations in blood glucose have been associated with increased craving and emotional instability in alcohol-dependent populations (Gold et al., 1985; Lieber, 2003). Preventing late-day metabolic decline appeared central to reducing anxiety-driven urges in this case, particularly given the patient's pattern of under-eating earlier in the day followed by heightened vulnerability in the evening.

Amino Acids as Symptom-Guided Support

Amino acids were used not as direct anti-addiction agents, but as targeted support for underlying neurobiological processes involved in mood regulation, stress tolerance, and craving vulnerability.

Chronic alcohol use is associated with dysregulation of neurotransmitter systems, including serotonergic, dopaminergic, and inhibitory pathways, which depend in part on the availability of amino acid precursors and micronutrient cofactors (Koob & Le Moal, 2001; Volkow et al., 2016).

From a biochemical perspective, amino acids such as 5-HTP (Birdsall, 1998) and L-glutamine (Rogers & Pelton, 1957; Williams, 1967) serve as precursors for neurotransmitters involved in mood and stress regulation, while compounds such as L-theanine may influence excitatory-inhibitory balance (Hidese et al., 2019). Nutritional psychiatry research further supports the role of nutrient availability in modulating brain function and emotional regulation (Jacka et al., 2017).

Within this framework, amino acids were used to support regulation during periods of increased vulnerability. The ability to interrupt cravings acutely with non-alcohol-based interventions, such as L-glutamine or L-theanine, provided an alternative pathway for managing distress in real time.

Clinical observations from earlier studies suggest that targeted nutrient and amino acid support may influence craving and emotional stability in individuals with substance use disorders, although high-quality controlled trials remain limited (Blum et al., 1988; Blum et al., 2011).

LIMITATIONS

As a single case report, causality cannot be established. Improvements likely reflected the combined effects of multiple concurrent interventions, including nutritional stabilization, amino acid and micronutrient support, psychotherapy, recovery-oriented social support, behavioral structure, and natural recovery processes. Laboratory confirmation of nutrient deficiencies was limited, and the relative contribution of any single intervention cannot be isolated. Additionally, perimenopausal hormonal changes may have contributed both to symptom burden and to subsequent improvements in sleep and stress regulation over time. Further case series and controlled studies are needed to better evaluate the role of orthomolecular and food-based approaches in supporting relapse prevention and psychological engagement in AUD.

CONCLUSION

In this patient, recovery appeared limited not by lack of motivation or insight, but by insufficient biological capacity to sustain change under stress. Targeted orthomolecular support appeared to stabilize physiology, reduce craving vulnerability, and enable psychological recovery to take hold.

These findings suggest that, for some individuals, addressing underlying physiological instability may be a necessary precondition for effective engagement in psychological and behavioral interventions. This case supports further exploration of orthomolecular and nutrition-based approaches as foundational components of AUD treatment.

ETHICS STATEMENT

This case report was prepared in accordance with the CARE Case Report Guidelines. Written informed consent was obtained from the patient for publication of this de-identified case report. All identifying information has been removed or altered to protect patient privacy.

CONFLICT OF INTEREST STATEMENT

The author declares no conflicts of interest related to this case report.

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AUTHOR CONTRIBUTIONS

Heather Dale conceived the clinical intervention, managed the case, and prepared the manuscript.

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