

# Recognizing and treating ketamine abuse

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The *N*-methyl-*D*-aspartate (NMDA) antagonist ketamine is a general-purpose anesthetic and Class III controlled substance that has a wide range of clinical applications. In addition to anesthesia, ketamine also is used for analgesia (acute and chronic pain) and critical care due to its receptor properties, which encompass adrenoceptors, purinergic, opioid, and cholinergic systems.<sup>1</sup> Because it has antidepressant effects, ketamine also is a novel treatment for treatment-resistant depression (TRD).

Physicians need to be aware, however, that many patients use illicit ketamine, either for recreational purposes or as self-treatment to control depressive symptoms. To help clinicians identify the signs of ketamine abuse, we discuss the adverse effects of illicit use, and suggest treatment approaches.

## Adverse effects of ketamine abuse

Ketamine can be consumed in various ways; snorting it in a powder form is a preferred route for recreational use.<sup>2</sup> The primary disadvantage of oral use is that it increases the likelihood of nausea and vomiting.<sup>2</sup>

While ketamine is generally safe in a supervised clinical setting, approximately 2.5 million individuals use various illicit forms of ketamine—which is known as Special K and by other names—in recreational settings (eg, dance clubs) where it might be used with other substances.<sup>3</sup> Alcohol, in particular, compounds the sedative effects of ketamine and can lead to death by overdose.

At a subanesthetic dose, ketamine can induce dissociative and/or transcenden-

tal states that are particularly attractive to those intrigued by mystical experiences, pronounced changes in perception, or euphoria.<sup>4</sup> High doses of ketamine—relative to a commonly used recreational dose—can produce a unique “K-hole” state in which a user is unable to control his/her body and could lose consciousness.<sup>5</sup> A K-hole state may trigger a cycle of delirium that warrants immediate clinical attention.<sup>3</sup>

Researchers have postulated that NMDA antagonism may negatively impact memory consolidation.<sup>3,6</sup> Even more troubling is the potential for systemic injuries because illicit ketamine use may contribute to ulcerative cystitis, severely disturbed kidney function (eg, hydronephrosis), or epigastric pain.<sup>3</sup> Chronic abuse tends to result in more systemic sequelae, affecting the bladder, kidneys, and heart. Adverse effects that require emergent care include blood in urine, changes in vision (eg, nystagmus), chest discomfort, labored breathing, agitation, seizures, and/or altered consciousness.<sup>6</sup>

## Treating ketamine abuse

Treatment should be tailored to the patient’s symptoms. If the patient pres-

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**Some patients with depressive symptoms may attempt to self-medicate using ketamine**

ents with “K-bladder” (ie, ketamine bladder syndrome), he/she may need surgical intervention or a cystectomy.<sup>4,7</sup> Therapeutic management of K-bladder entails recognizing bladder symptoms that are specific to ketamine use, such as interstitial or ulcerative cystitis and lower urinary tract symptoms.<sup>7</sup> Clinicians should monitor patients for increased voiding episodes during the day, voiding urgency, or a general sense of bladder fullness. Patients with K-bladder also may complain of suprapubic pain or blood in the urine.<sup>7</sup>

Consider referring patients to an individualized, ketamine-specific rehabilitation program that is modeled after other substance-specific rehabilitation programs. It is critical to address withdrawal symptoms (eg, anorexia, fatigue, tremors, chills, tachycardia, nightmares, etc.). Patients undergoing ketamine withdrawal may develop anxiety and depression, with or without suicidal ideation, that might persist during a 4- to 5-day withdrawal period.<sup>8</sup>

**‘Self-medicating’ ketamine users**

Clinicians need to be particularly vigilant for situations in which a patient has used ketamine in an attempt to control his/her depressive symptoms. Some researchers have described ketamine as a revolutionary drug for TRD, and it is reasonable to suspect that some patients with depressive symptoms may have consulted Internet sources to learn how to self-medicate using ketamine. Patients who have consumed smaller doses of ket-

amine recreationally may have developed a tolerance in which the receptors are no longer responsive to the effects at that dose, and therefore might not respond when given ketamine in a clinical setting. Proper history taking and patient education are essential for these users, and clinicians may need to develop a personalized therapeutic plan for ketamine administration. If, on the other hand, a patient has a history of chronic ketamine use (perhaps at high doses), depression may occur secondary to this type of ketamine abuse. For such patients, clinicians should explore alternative treatment modalities, such as transcranial magnetic stimulation.

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