Extromethorphan (DXM) is commonly found in over-the-counter (OTC) cold and cough preparations. When used at the therapeutic doses DXM has cough-suppressant properties through its action on the medulla. However, OTC preparations containing DXM are being increasingly used recreationally for the drug’s psychoactive effects, a practice referred to as “robotripping.” Such use can result in a toxidrome of delirium with agitation, paranoia, and hallucinations. Residents need to be able to recognize the signs of DXM abuse and manage its potentially serious complications.

**How DXM works**

DXM has a wide therapeutic window. A typical therapeutic dose for cough is up to 120 mg/d. The most common adverse effects are mild (fever, diaphoresis, dizziness, nausea). At higher dosages, it acts as a nonselective serotonin reuptake inhibitor, a sigma-1 receptor agonist, and an N-methyl-D-aspartate (NMDA) receptor antagonist. DXM produces psychoactive effects through its active metabolite, dextrorphan, which has high affinity for NMDA. In this way, it can produce dissociative and stimulant effects. Although the amount of DXM in commercially available cold and cough preparations is modest, instructions for extraction and purification are readily available on the Internet.

Adverse effects include hallucinations, disorientation, mania, and aggression with delusions of supernatural abilities and insensitivity to pain; these effects are similar to those produced by phencyclidine (PCP). Physiologically, diaphoresis, hyperthermia, and tachycardia are often observed. These presentations carry a significant risk of mortality, and appropriate recognition and management is needed.

**4 Phases of intoxication**

DXM users have described 4 progressive behavioral phases that vary with dosage. First, at 1.5 to 2.5 mg/kg, users report stimulating effects with perceptual alterations similar to those produced by 3,4-methylene-dioxymethamphetamine (“ecstasy”). The second phase, reached at 2.5 to 7.5 mg/kg, is similar to alcohol and marijuana intoxication but includes more pronounced dysfunction in motor, cognitive, and perceptual skills, and perhaps visual hallucinations. The third phase, noted at 7.5 to 15 mg/kg, resembles ketamine intoxication, with strong dissociation and hallucinations. At greater doses, out-of-body, trance-like experiences may occur. Delirious misperceptions often lead to violent behavior and limited perception of pain. Users may experience a long course of any of these phases, with presentations lasting for up to 1 to 2 weeks after discontinuing use.

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Management is mainly supportive
Early recognition of DXM use is essential for treatment. Unfortunately, without collateral reports, this can be challenging because specialized toxicology screens are needed to detect DXM. Basic screens sometimes show a false positive for PCP. Take an inventory of all substances in the patient’s possession, either by examining the patient’s belongings or by obtaining collateral information from the patient’s family or friends.

Supportive care should be implemented, with a primary goal of controlling agitation. Short-acting benzodiazepines are helpful. Low-dose, short-term antipsychotics have shown benefit when hallucinations and paranoia are prominent. Decreasing stimulation and avoiding physical restraints while attempting to control aggression and psychosis with these medications is recommended. Using physical restraints on an individual who is in a state of agitated delirium can lead to severe injuries, cardiac and respiratory arrest, and death.

Patients typically experience rapid and complete remission of symptoms after discontinuing DXM use. However, evidence suggests DXM users can develop tolerance as well as psychological and physiological dependence. DXM withdrawal can be quite protracted and may include anxiety, dysphoria, insomnia, and suicidality.

References