Lessons from practice

Severe GHB withdrawal delirium managed with dexmedetomidine

Clinical record

A 23-year-old woman was sent to our emergency department with severe agitation 12 hours after admission to a private detoxification facility. She had taken γ-hydroxybutyrate (GHB) and methamphetamine that day, and had reportedly presented to the detoxification unit well.

The woman had been dependent on GHB and γ-butyrolactone (GBL) for 2.5 years, and had misused methamphetamine and benzodiazepines. She took up to 36 mL GHB/GBL, 3 points (0.3 g) of methamphetamine, and 25 mg diazepam daily. Her other diagnoses included depression treated with venlafaxine, and mild attention deficit disorder that had previously been treated with methylphenidate.

At the emergency department, she was agitated and responding to hallucinations. She was tremulous, diaphoretic, and tachycardic. She was inattentive, unable to maintain reasonable conversation, and displayed elements of paranoia and thought disorder. She had to be physically restrained. The delirium work-up suggested no alternative diagnoses.

The patient was administered 65 mg diazepam, 50 mg quetiapine and 10 mg olanzapine during the first 24 hours, without marked benefit. Upon transfer to the intensive care unit, dexmedetomidine infusion was commenced. The infusion rate was titrated (1 to 0.2 mg/h) to limit dexmedetomidine-induced bradycardia and hypotension. Ventilation support was not required. She also received 30–55 mg diazepam each day. By Day 5, the patient was still agitated and responding to visual and auditory hallucinations, although to a lesser degree, and the dexmedetomidine infusion was stopped. She continued to require one-to-one nursing care for verbal re-direction, with good effect, as well as diazepam when her agitation increased.

After 2 weeks, most of her symptoms had subsided and the patient was no longer delirious. She was discharged to a private rehabilitation facility.

She was still abstinent from GHB/GBL at one-year follow-up.

Early features of GHB withdrawal resemble those of alcohol withdrawal, including autonomic instability, tremor, anxiety, agitation and altered sleep. As with our patient, severe withdrawal can include acute delirium that requires intensive care. Given its short half-life, withdrawal symptoms can start within hours of the last use, and are thought to be due to the loss of GHB-mediated inhibitory effects in the central nervous system, resulting in a hyper-excitatory state. Withdrawal can last 3–21 days.

Benzodiazepines (GABA<sub>Α</sub> receptor agonists) have been the mainstay of the pharmacotherapy for GHB withdrawal, and large quantities are usually required. Benzodiazepine-resistant patients have been reported, necessitating the use of other sedative agents, including propofol and baclofen. We report the application of dexmedetomidine, a short-acting α<sub>2</sub>-adrenoceptor agonist, for managing GHB withdrawal. Dexmedetomidine has the advantage of producing relatively limited respiratory depression and a qualitatively different sedation to benzodiazepines; it reduces the prevalence of drug withdrawal.


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delirium, and possesses analgesic effects, unlike benzodiazepines. However, the duration of intensive care and overall hospital length of stay are similar to benzodiazepine management.

Early recognition and prompt implementation of appropriate care are crucial for preventing the more severe complications of acute withdrawal. Dexmedetomidine sedation may help facilitate a less invasive and safer approach to intensive care management of delirium secondary to drug withdrawal.

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