



Gamma-Hydroxybutyrate (GHB) Intoxication Requiring ICU Admission for Acute Toxic Encephalopathy

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BACKGROUND

- GHB is used as the treatment for narcolepsy and in some countries for alcohol and opiate dependence.¹
- GHB acts on the GABA-B and GHB receptor in the brain which causes sedative and excitatory effects on the CNS leading to a combination of euphoria and respiratory depression.
- It is commonly abused and used recreationally.
- Along with ketamine and flunitrazepam, GHB is one of the most common agents used in drug-facilitated sexual assaults.

CASE PRESENTATION

Admission

- A 26 year old male was brought by EMS to the emergency department for altered mental status.
- Patient had been drinking the night prior and ingested Adderall.
- EMS reported that the patient possibly had seizures, and seemed to be in post ictal state when they arrived.

Medical History

- No significant past medical history.

Physical Exam

- Vital signs stable with mild hypoxia noted on room air.
- Respiratory distress with rhonchorous breathing, notable desaturations into the 70-80s despite high flow oxygen; obtunded, diffusely diaphoretic, vomit in mouth.

Laboratory Data/Radiographic

- Total CK: 825.0.
- Arterial Blood Gas: pH 7.280, pCO2 55.0, bicarb 25.8.
- Urine Toxicology: +amphetamines.
- Urine Gamma-Hydroxybutyric Acid (GHB): 1400 mcg/mL (Report Limit: 50).
- Urine GHB Creatinine Corrected: 790 mg/g Creatinine (Report Limit: 28).
- CT Cranial was unremarkable.
- CXR and CTA Chest suspicious for aspiration pneumonia.

CLINICAL COURSE

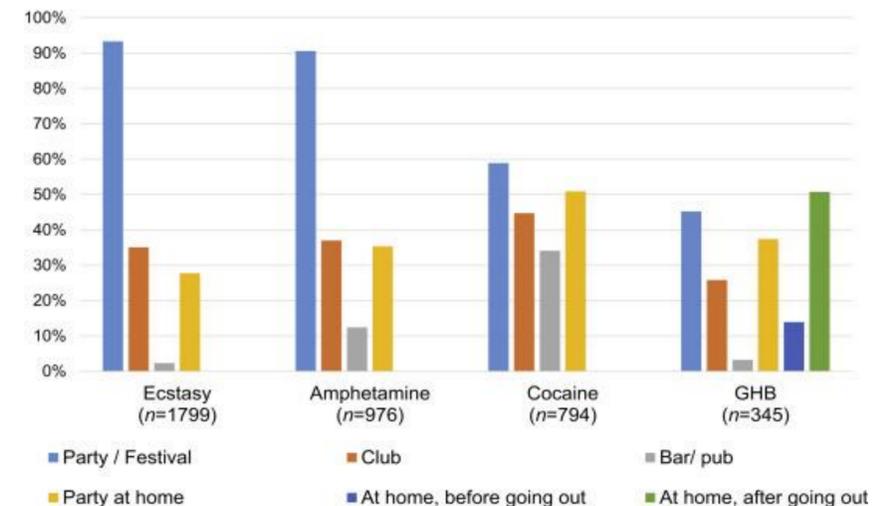
- In the ED, the patient was hypoxic requiring intubation and sedation.
- He was admitted to the ICU. He had two episodes of severe agitation and was combative, requiring restraints and midazolam.
- His mentation and agitation gradually improved over the proceeding 24 hours and he was successfully extubated.

DISCUSSION

- GHB effects on the CNS at lower doses activate dopamine release, causing a euphoric effect.
- At higher levels, it inhibits dopamine release via agonist effects on GABA-B receptors, leading to neuronal-inhibition and central nervous system (CNS) depression.
- Patients with GHB toxicity will present to the ED altered, bradycardic, and hypotensive. This can be followed by acute agitation and combativeness. Patients may also have amnesia, myoclonic activity, and absence-like seizures.
- Equally, acute GHB withdrawals can progress to agitation, psychosis, and autonomic dysfunction requiring ICU admission.
- Withdrawal symptoms can start within 1-6 hours after the last dose, and last up to 5-15 days.
- It can present as tachycardia, hypertension, paranoid delusions, hallucinations, and rapid fluctuations in sensorium.
- Typically treated with benzodiazepines but in severe cases that are refractory, phenobarbital has shown efficacy in controlling behavioral, autonomic, and psychiatric symptoms.
- GHB testing is generally performed on urine samples. Depending on the ingested dose and the patient's renal function GHB may be detected in the urine up to 24 hours after ingestion; in patients with normal renal function GHB is often cleared from the body within 4-6 hours and negative urine testing is not unusual if performed later in the patient's presentation.

CONCLUSIONS

- Both GHB toxicity and withdrawals can be life threatening.
- GHB testing is rarely performed during standard urine drug screening. Intoxication should be considered in the differential with patients who presents with acute encephalopathy, substance use, and a negative rapid urine drug screen.
- Dedicated urine testing may reveal the presence of GHB.
- It's important to provide supportive care not only during the initial toxicity but also after, closely monitoring for withdrawal symptoms.
- Patients should be admitted to the ICU once GHB toxicity is suspected as their respiratory function may acutely decompensate requiring intubation.



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