GHB in Your Patients: Recognizing The Characteristics

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Gamma-hydroxybutyrate (GHB) is a well known scheduled drug used both therapeutically and recreationally. It is an analogue of γ-aminobutyric acid (GABA), the main inhibitory neurotransmitter in the central nervous system (CNS). GHB was initially introduced in the 1960s as a general anesthetic, then was used as a dietary supplement for weight training due to its reputed effect as a growth hormone stimulant. In the 1990s, GHB gained popularity in the rave scene, in addition to its use as a date-rape drug. This drug has caused fatalities, been implicated in impaired driving cases and also resulted in ED visits requiring invasive interventions. More recently, GHB received approval as a prescription drug (sodium oxybate) for treatment of narcolepsy.

Due to the strict regulation of GHB and its precursors in the US, GHB use has been on the decline in the last three to four years. However, GHB is still a significant issue in Europe and Australia.

Effects

GHB is only available as a liquid, as are GHB precursors (butanediol and γ-butyrolactone). It is rapidly absorbed and peak blood levels occur 15 to 45 minutes after ingestion and the “therapeutic window” for this toxin is narrow. Due to

Lisa’s case

Lisa, 22, is brought in by paramedics to the ED. Lisa was at a rave party and was found by her friends collapsed on the dance floor. She had a Glasgow Coma Scale score of 3. When stimulated, she had brief episodes of combative behaviour. Because of this the paramedics had a difficult time intubating her in the field and had to administer IV valium. The paramedics also made note of emesis at the scene. Her vital signs are:

- Pulse: 56 bpm
- BP: 95/60 mmHg
- Respiratory rate: 14 breaths/minute on the ventilator, O₂ saturation was 100% on 100% oxygen
- Temperature: 36˚C, glucometer reading of 5.5 mmol

ECG showed a normal sinus rhythm. Physical exam was significant for myoclonic jerks with otherwise flaccid paralysis.

All bloodwork revealed no abnormalities. An unenhanced CT scan of her head was also normal. She did not require sedation despite remaining intubated.

Two hours after presentation to the ED, Lisa awakes suddenly and pulls out her endotracheal tube and IV lines. She has a completely normal physical exam and mental status. She admits to use of ethanol and “liquid X.” She leaves the department against medical advice.

1. What is the most likely nature of “liquid X”?
2. Which characteristics are typical of this toxin in this case?
3. Should you worry that she has left against medical advice?
the fact that it comes in liquid form, it is difficult for the user to be aware of the exact dose they have taken, since drug concentrations differ markedly. In addition, this liquid can be masked in an ethanolic beverage for the purpose of sexual assault.

GHB is used recreationally for its euphoric and sexual enhancement effects. It is mostly used in conjunction with other recreational drugs/drugs of abuse, such as ethanol, cocaine, methylenedioxymethamphetamine (MDMA/ecstasy) or ketamine.

The main clinical effect of GHB is CNS depression due to its activation of the GABA receptor. This is potentiated by the frequent co-ingestion of other CNS depressants, most notably ethanol. Respiratory depression also occurs commonly and can result in respiratory arrest. For both the profound CNS and respiratory depression, endotracheal intubation is often required. The characteristic findings of this intoxication are deep coma with brief consciousness upon stimulation and abrupt reversal of these symptoms within a few hours of presentation to a healthcare facility. Patients typically become combative and pull out all medical devices upon awakening. If the coma persists beyond four to six hours, another reason for the clinical presentation must be sought.

Other clinical features of GHB intoxication include:

- seizures,
- bradycardia and
- mild hypothermia.

A withdrawal syndrome due to chronic use of GHB has also been described and resembles that of withdrawal from ethanol or benzodiazepines. Tremor, agitation, hallucinations, hypertension and tachycardia may occur.

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**Treatment**

When a patient presents to the ED with coma, our approach is always the same: airway, breathing, circulation followed by resuscitation and further evaluation as required. GHB often results in patients with Glasgow Coma Scale (GCS) below eight, which requires definitive airway management with endotracheal intubation. Otherwise, the usual supportive care with IV fluids and possibly sedation are all that is necessary. Decontamination with activated charcoal is not recommended for a number of reasons:

- risk of aspiration,
- small amounts of drug ingested in the context of recreational use and
- rapid absorption of the liquid drug.

Antidotes, such as naloxone, flumazenil and physostigmine, have not been shown to be effective and therefore are not recommended.
If there is co-ingestion of MDMA or cocaine, then other serious complications requiring intervention may co-exist, such as:

- hyperthermia,
- rhabdomyolysis,
- myocardial ischemia,
- intra-cerebral edema or bleed,
- hyponatremia and
- seizures may occur.

If patients present with signs of infection, trauma or signs indicating other causes for coma these must be pursued immediately despite a history of substance abuse.

**Conclusion**

GHB is a potent CNS depressant which can result in serious sequelae. The rapid onset of effect and narrow therapeutic window increase the likelihood of ingesting more than intended. It is frequently co-ingested with ethanol, stimulants or ketamine, further increasing the potential for miscalculation. Although GHB use is declining, recognizing the characteristics of toxicity is important for the clinician.

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**Resources**