Toxicology Case: A Comatose Patient with Wide Complex Tachycardia in Emergency Department

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Introduction

Coma is a deep state of unconsciousness during which an individual is unable to react to his environment. Stupor refers to a state of severely impaired arousal with some purposeful responsiveness to vigorous stimuli.

The comatose patient is a common presentation to the ED, and can be a diagnostic and treatment challenge for the emergency department physician. Coma can occur as a result of a variety of reasons including, neurologic, metabolic, toxicological and other conditions.

Case Presentation

A 17-year-old comatose female was brought to the Emergency Department by ambulance. According to paramedics, she had been found in her home in a comatose state. No additional history was provided.

Physical examination demonstrated a patient in a comatose state. The pupils were narrowed and there was slow light response. Blood pressure was 100/60 mm Hg, and pulse was regular at 125/min. Her core temperature was rectally measured at 36.6 C. Oxygen saturation was measured in room air at 92% and respiratory rate was 20/min. After administering oxygen at 5
liters/min via face-mask, the saturation rose to 95%.

The patient was without obvious pallor, cyanosis or jaundice. There were no outward signs of physical violence. There was no sign of her having bit her tongue or lost control of bowel or bladder. Neurologic examination was without meningeal signs of focal neurologic deficit. Auscultation of the lungs and the heart were normal, as was the abdominal exam.

Initial management and investigations

The patient’s blood glucose test was within normal range. Because of narrowed pupils in our patient she was given Naloxone 0.8 mg without any response. Intramuscular Thiamine (vitamin B1) 100 mg was administered.

Laboratory examinations revealed a negative Beta-HCG, and normal liver and kidney function tests. Blood coaguability and D-Dimer test results were within the normal range. Blood level of ammonia was also normal.

Urinary toxicology screen including amphetamines, opiates, heroin, cannabinoids and benzodiazepines was negative.

Arterial blood gases showed: pH 7.25, PO2 60, PCO2 39, bicarbonate 18, oxygen saturation 90%. Level of CoHGB was nontoxic.

ECG showed normal sinus rhythm at approximately 125/min with borderline widening of the QRS-complex without signs of ischemia.

We intubated the patient. Gastric lavage was performed without evidence of any substance that had been ingested.

At this point, the patient’s family arrived in the Emergency Department and additional information was provided. Collateral information revealed that the patient had been suffering from depression and was undergoing pharmaceutical treatment for this condition.

A Brain CT was ordered. While waiting for transfer to the CT unit, the patient’s hemodynamic status precipitously deteriorated.

The cardiac monitor demonstrated wide complex tachycardia on her monitor. In light of what just happened, the patient was defibrillated twice at 200 J. In addition, she was given IV Lidocaine 200 mg. There was no response to any of the above treatment.

Given the new information and the patient’s current critical hemodynamic situation, we administered 2 ampoules of IV bicarbonate in order to counter the effects of a possible tricyclic antidepressant overdose. The patient’s hemodynamic condition improved with a return to normal sinus rhythm at approximately 100/min with gradual disappearance of QRS widening.

Additional information from the family revealed that the patient had ingested 40 tablets of Elatrol (Amitriptyline Hcl) 25 mg/tablet, in a suicide attempt approximately 6 hours earlier.

Outcome

The patient was moved from the Emergency Department to the Intensive Care Unit in a hemodynamically stable condition with a diagnosis of coma and wide complex tachycardia due to tricyclic antidepressant overdose. After receiving symptomatic treatment with IV bicarbonate supplementation, the patient’s condition continued to improve. The next morning, the patient was extubated and fully alert.

Discussion

Tricyclic antidepressants (TCAs) are used in the treatment of depression, chronic pain and enuresis. Patients with depression and those with chronic pain are at high risk for abuse, misuse and overdosing of these drugs. The juvenile population being treated for enuresis is at risk for accidental overdose. The first lethal reported overdose occurred in 1959 when TCAs were used widely to treat depression. Although they are currently less popular for the treatment of depression, TCAs still represent a significant proportion of lethal overdoses. The primary complications of ingestion result from cardiovascular and central nervous system (CNS) toxicity. Early recognition and specific appropriate treatment are necessary to achieve good clinical outcome.
The toxic effects on the myocardium are related to the blocking of fast sodium channels, in a similar mechanism to that of type IA antiarrhythmics (e.g., quinidine). The result is a slowing myocardial depolarization that leads to arrhythmia, myocardial depression and hypotension. Hypotension also results from peripheral alpha-adrenergic blockade, which causes vascular dilation.

CNS toxicity results from the anticholinergic effects and direct inhibition of bioamine reuptake. An excitation syndrome is the initial result and manifests as confusion, hallucinations, ataxia, seizures and coma. The effects on the pulmonary system include pulmonary edema, adult respiratory distress syndrome and aspiration pneumonitis. The anticholinergic effects of TCAs cause a slowing of the gastrointestinal (GI) system, which results in delayed gastric emptying, decreased motility and prolonged transit time.

Onset of symptoms after tricyclic antidepressant overdose typically occurs within two hours, and life-threatening complications occur within six hours. A usual therapeutic dose of any TCA is 2-4 mg/kg/d. A potentially lethal dose is 10-15 mg/kg.

No correlation exists between TCA blood level and symptoms. Routine screening for other potentially treatable toxins is recommended (e.g., aspirin, acetaminophen). Additionally, measurements of electrolytes, BUN, creatinine and anion gap must be obtained. An arterial blood gas (ABG) test should be performed for management and/or monitoring of treatment.

Electrocardiogram right-axis deviation of terminal 40 milliseconds of QRS in the frontal plane (limb leads) is a sign of TCA exposure and a possible predictor of toxicity. An R wave in AVR more than or equal to 3 mm is an ECG variable that may predict adverse outcomes.

A QRS less than 100 milliseconds is a negligible risk for seizures or arrhythmias; more than 160 milliseconds is a high risk.

As a result of tricyclic antidepressant overdose, AV blocks and bundle branch block may be observed.

### Emergency Department Essentials

- Perform baseline and serial ECGs to monitor the QRS duration if TCA ingestion is known or suspected.
- Intubate if coma or significantly depressed mental status is present. Benzodiazepines and barbiturates are the preferred induction agents; however, avoid barbiturates if hypotension is present. Caution should be exercised when hyperventilating a patient during bicarbonate administration, as this may produce severe alkalemia.
- Hemodialysis and hemoperfusion have not been shown to be effective.
- Alkalemia has been shown to protect against and treat dysrhythmias.
- Perform orogastric lavage if ingestion is known to be lethal. Intubate before lavage to decrease the risk of aspiration.
- Administer activated charcoal when appropriate.
- IV Sodium bicarbonate is used to normalize serum pH. First-line therapy for QRS >100ms or if dysrhythmias are present. Doses or IV drip may be administered with a maximum pH of 7.5. IV Sodium bicarbonate 1-2 mEq/kg bolus IV should be administered; IV drip of 3 amps of sodium bicarbonate in 1 L of D5W to maintain a pH of 7.45-7.5.
- Lidocaine is a class IB antiarrhythmic that increases the electrical stimulation threshold of ventricle, suppressing automaticity of conduction through tissue. It is a second-line drug for dysrhythmias. Lidocaine is administered as 1-1.5 mg/kg IV bolus, which can be repeated to a maximum of 3 mg/kg; with a maintenance drip of 1-4 mg/min.
- IV Diazepam is an anticonvulsive that depresses all levels of CNS (e.g., limbic and reticular formation), possibly by increasing the GABA activity. Another benzodiazepine agent, IV lorazepam can also be used.
- Phenobarbital is used for seizures not responding to benzodiazepine. Loading dose is 15-20 mg/kg IV at 25-30 mg/min. It is not to exceed 300-800 mg.
- Flumazenil is potentially hazardous, as it reverses the protection from seizures in mixed TCA – benzodiazepine overdose.
Conclusions

The above case emphasizes the importance of appropriate history taking from family members and high clinical suspicion in cases of tricyclic antidepressant overdose. In cases of coma with metabolic acidosis and cardiac dysrhythmia with QRS complex widening, the emergency physician must keep in mind the possibility of tricyclic antidepressant overdose. Intravenous bicarbonate in this scenario can be life-saving. Constant and close monitoring and reevaluation of the patient’s condition is essential for successful management of TCA overdose.

References


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