

EM CASE OF THE WEEK.

BROWARD HEALTH MEDICAL CENTER
DEPARTMENT OF EMERGENCY MEDICINE



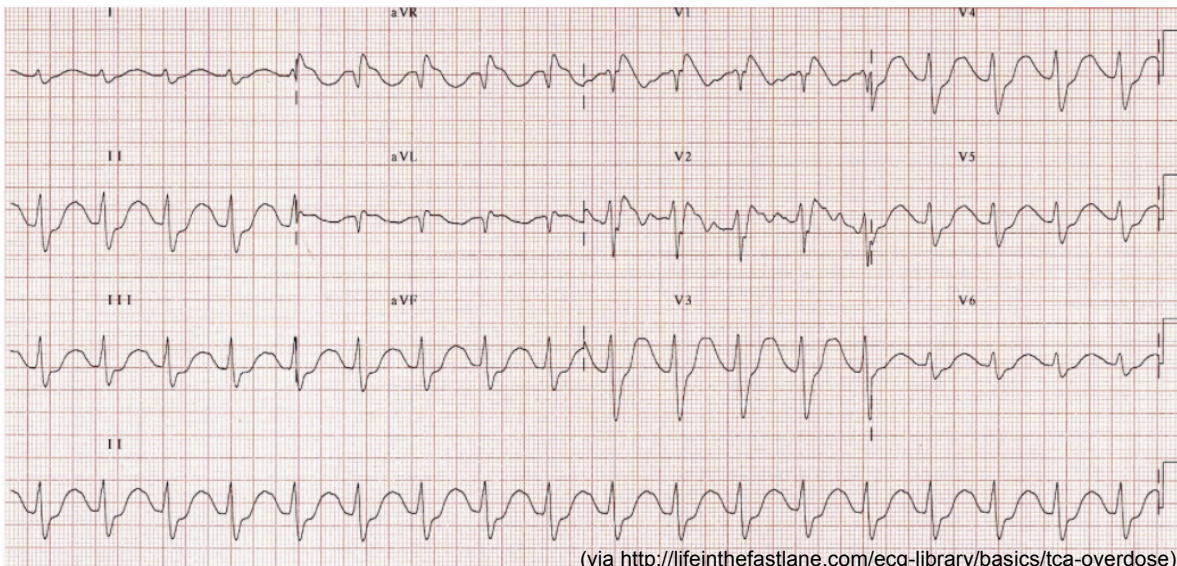
Care Warriors

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Tricyclic Antidepressant (TCA) Toxicity

A 28-year-old female with a history of depression is brought to the ED by her husband who says she has been acting confused. The husband states she has been very depressed lately and he came home this evening to find her acting confused and agitated. He also noticed her newly filled antidepressant bottle empty on the floor. The patient appears drowsy, confused, and somewhat agitated, stating, "the light is dripping down the wall." She has a temperature of 100.2 F, vitals show a pulse of 112 bpm and a BP of 92/66 mmHg. On physical exam, the patient appears flushed with dry oral mucosa. Her pupils are dilated and respond poorly to light. Her exam is otherwise unremarkable. Her EKG upon arrival is shown below:



(via <http://lifeinthefastlane.com/ecg-library/basics/tca-overdose/>)

Which of the following is the best initial treatment for this patient's condition?

- A. Magnesium, 1g IV over 15min
- B. 50 mEq NaHCO₃ IV push
- C. Synchronized cardioversion
- D. Lidocaine, 1.5mg/kg IV
- E. 150 mEq NaHCO₃ in D5W IV
- F. Amiodarone, 150mg IV

EM Case of the Week is a weekly "pop quiz" for ED staff.

The goal is to educate all ED personnel by sharing common pearls and pitfalls involving the care of ED patients. We intend on providing better patient care through better education for our nurses and staff.

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The correct answer is **B**, rapid infusion of 50 mEq sodium bicarbonate.

Discussion

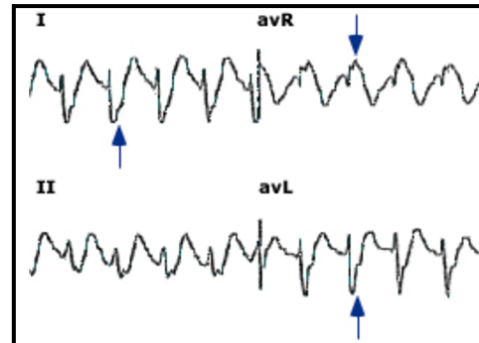
Tricyclic antidepressants (TCAs) act largely by blocking the reuptake of serotonin and norepinephrine. To a lesser degree, they block fast sodium channels and antagonize acetylcholine, alpha-1, histamine H1, and GABA receptors. TCAs have largely been replaced by selective serotonin reuptake inhibitors (SSRIs) for the treatment of depression; however, they are still occasionally used to treat bedwetting, neuropathic pain, and treatment-resistant depression.

Patients who overdose on TCAs typically present with a constellation of symptoms roughly 2-5 hours after ingestion. These include CNS symptoms of drowsiness, confusion, and delirium, as well as anticholinergic signs of blurry vision, warm/dry skin, dry mouth, urinary retention, and tachycardia. The antagonism of GABA receptors can result in a lower seizure threshold and the antagonism of alpha-1 receptors can lead to hypotension. The antagonism of fast sodium channels in the His-Purkinje system leads to significant cardiac conduction abnormalities including a prolonged QRS complex and prolonged QT/QTc interval.

Significant cardiovascular and CNS toxicities with rapid deterioration will begin to develop after ingestion of 10-20 mg/kg of a TCA.

Diagnosis

TCA toxicity is diagnosed based on a combination of high suspicion for TCA ingestion, clinical symptoms consistent with TCA overdose, and predictable ECG changes. While serum TCA levels can be measured, they are not available in a timely fashion and are not predictable of toxicity – therefore, their use is limited in the acute setting and does not guide therapy.



(via <http://www.uptodate.com/tricyclic-antidepressant-poisoning>)

The ECG changes that are found in TCA toxicity consist of the following:

1. **QRS duration >100 ms**
2. **Abnormal QRS morphology**
 - a. Deep, slurred S wave in I, aVL
3. **Abnormal R and S waves in avR**
 - a. R wave > 3mm + R/S ratio > 0.7

The degree of QRS widening can be correlated with the severity of the toxicity¹. A QRS >100 ms predicts a 25% chance of seizure development and a QRS >160 ms predicts a 50% chance of ventricular arrhythmia.

Treatment

Treatment of TCA overdose begins with the ABCs. Intubate the patient as indicated to protect the airway. If QRS is >100, rapidly administer 2-3 mEq/kg sodium bicarbonate IV. The QRS can be observed to narrow in real-time on ECG. If QRS is responsive to initial sodium bicarbonate administration, begin continuous infusion of 150 mEq sodium bicarbonate in 1L D5W at 250 mL/hr. If the QRS is not responsive, 1g IV magnesium or 1.5 mg/Kg Lidocaine can be used. The hypotension can be refractory to fluids and should be supported with vasopressors if needed; norepinephrine is preferred². Treat seizures with benzodiazepines. Avoid physostigmine due to the risk of asystole³.

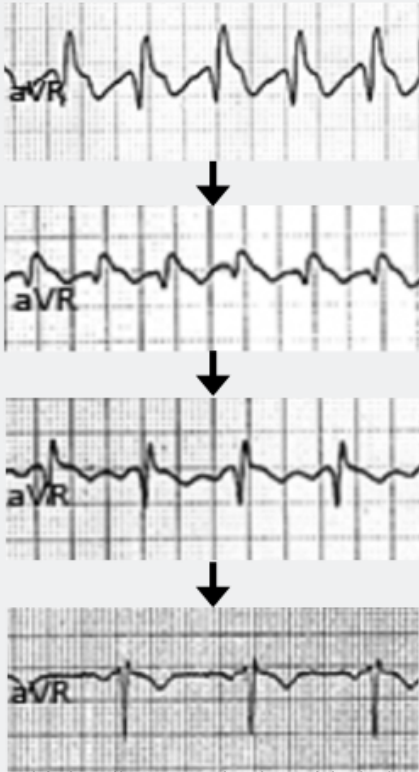
Check serial ABGs with a goal pH of 7.5-7.55. Repeat EKGs hourly until resolution of conduction abnormalities. Also consider testing for coingestion of other drugs.

For a list of educational lectures, grand rounds, workshops, and didactics please visit BrowardER.com and click on the **“Conference”** link.

All are welcome to attend!

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ECG Progression



This image illustrates the progression of TCA toxicity on lead aVR followed by treatment with bicarbonate. The first box shows early toxicity with a widened QRS of 144 ms, and characteristic changes in aVR. The second box shows the progression to severe TCA toxicity with a QRS of 160 ms. The third box is after an initial treatment with bicarbonate, which results in a narrowing of the QRS to 108 ms. The final box shows the resolution of the toxin-related cardiac conduction abnormalities and a normal QRS of 88.



ABOUT THE AUTHOR

This month's case was written by Brad Parrish. Brad is a 4th year medical student from NSU-COM. He did his Emergency Medicine rotation at BHMC in September 2016. Brad plans on pursuing a career in Emergency Medicine after graduation.

Take Home Points

- Clinical features of TCA overdose: Confusion, delirium, seizures, anticholinergic effects (dilated pupils, flushed skin, dry mouth, urinary retention), tachycardia, and hypotension.
- EKG findings: Tachycardia, widened QRS, abnormal QRS morphology, and abnormal R and S waves in aVR.
- Initial treatment with 1-2 mEq of sodium bicarbonate IV push and assess for QRS narrowing. If responsive, begin 150 mEq bicarbonate in D5W maintenance.
- If QRS non-responsive to bicarbonate, administer 1 mg magnesium or 1.5 mg/Kg lidocaine.
- Maintain blood pressure with IVF and vasopressors as needed, alpha-adrenergic agonists are best.
- Avoid the use of physostigmine

REFERENCES

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3. Salhanick SD. Tricyclic antidepressant poisoning. In: *UpToDate*, Basow, DS (Ed), UpToDate, Waltham, MA, 2016.
4. Tran TP, Panacek EA, Rhee KJ, Foulke GE. Response to dopamine vs norepinephrine in tricyclic antidepressant-induced hypotension. *Acad Emerg Med.* 1997;4(9):864.