

EMQN Sample Case and Structure

Updated version to increase rigor for publication and DOI assignment

STRUCTURE:

Abstract- less than 150 words

Intro

Case- case info

Discussion- what you want to teach

1. clinical guidelines
2. care standards
3. therapy
4. follow-up/care coordination
5. link to guidelines/cpg (if applicable)

Conclusion

Quiz

Must have sources--No more than 5 sources, cited AMA style

Between 1500-2500 words.

Label all images/tables, include explanation as necessary

3-5 (hitting high yield points) with explanations.

No true or false questions.

No "all of the above" choices

Please include why incorrect choices are incorrect in the explanation

All you need to submit to me is a word doc with the case/quiz/resources, etc

I'll put the email together/send

I'll attach any attachments you need to go with the case as applicable

Thank you all again!!

Bobby

SAMPLE:

Abstract

A 17-year-old female suffered intentional overdose of amitriptyline. Arriving with diminished responsiveness, tachycardia, and a widened QRS with a prolonged QTc, the patient exhibited classic

anticholinergic and cardiotoxic toxidromes. Treatment required aggressive sodium bicarbonate boluses and infusions to overcome rapid cardiac sodium channel blockade, alongside intravenous magnesium sulfate for QTc prolongation and benzodiazepines for generalized tonic-clonic seizures. The patient necessitated intubation for airway protection but was successfully extubated and transferred to psychiatric care by hospital day four. This case underscores the critical recognition of tricyclic antidepressant (TCA) toxicity, underlying electrophysiological pathophysiology, and the time-sensitive stabilization interventions essential for managing life-threatening dysrhythmias and neurological complications.

Intro

Imipramine was the first tricyclic antidepressant (TCA) introduced in the late 1950s, serving as the major pharmacologic treatment for depression until safer medications emerged in the late 1980s. Recently, there has been a resurgence in TCA availability as these medications are increasingly prescribed for chronic pain, obsessive-compulsive disorder (OCD), migraines, and insomnia. Because of this increased availability, clinicians must maintain a high index of suspicion and remain proficient in identifying and managing acute TCA toxicity.

Case

A 17-year-old female presented after intentionally overdosing on approximately 20 tablets of her prescribed amitriptyline around one hour prior to arrival. The patient's mother activated EMS upon noticing abnormal behavior and finding the empty medication bottle.

On presentation, the patient was minimally responsive with a Glasgow Coma Scale (GCS) of 5. Initial vital signs demonstrated a heart rate of 126, a blood pressure of 107/72, an oxygen saturation of 100% on room air, a temperature of 98.2F, and a respiratory rate of 18. The physical exam revealed dry mucous membranes, diminished bowel sounds, normal extremity tone, unremarkable patellar reflexes without clonus, and 7mm sluggish pupils bilaterally.

Laboratory results showed normal liver function tests, normal electrolytes, undetectably low acetaminophen and salicylate levels, an ethanol level of 76, and a point-of-care glucose of 105. The initial ECG (See Figure 1) revealed sinus tachycardia with a QRS of 104 msec and a markedly prolonged QTc of 573 msec.

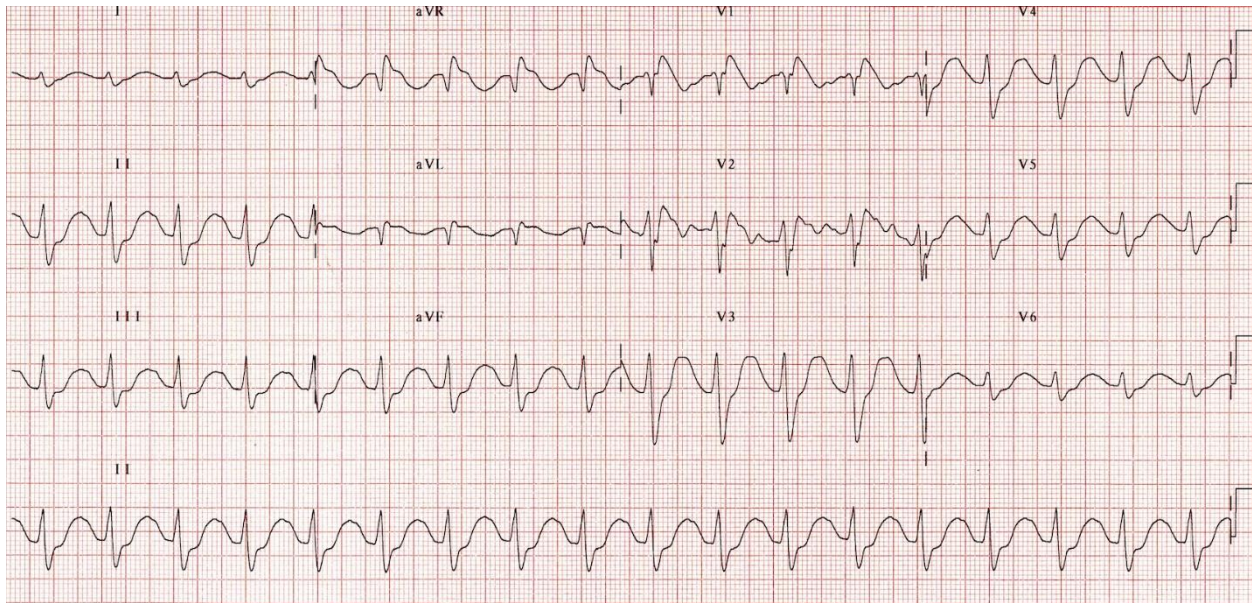


Figure 1: Initial ECG after acute TCA overdose

The patient's clinical status deteriorated with increasing tachycardia to a rate of 141, a QRS widening to 130 msec, and a QTc of 545 msec. Interventions began with two 50 mEq ampules of sodium bicarbonate, which improved the QRS to 118 msec. A third ampule of sodium bicarbonate further narrowed the QRS to 110 msec, after which a continuous bicarbonate infusion was initiated at twice the maintenance rate. The patient was given 2g of intravenous magnesium sulfate to treat the QTc prolongation. Following a brief generalized tonic-clonic seizure that was aborted with 4mg of IV lorazepam, the patient was intubated for airway protection and started on a propofol infusion for sedation.

The tachycardia and ECG intervals slowly improved, allowing the bicarbonate infusion to be discontinued on hospital day 2. The patient was successfully extubated on hospital day 4 and subsequently transferred to an inpatient psychiatric unit in good condition.

Discussion

The primary pathophysiology of acute TCA overdose is cardiotoxicity caused by a slowed recovery from the inactivation of fast cardiac sodium channels during phase 0 depolarization within the distal His-Purkinje system and ventricular myocardium. Cardiac sodium channel blockade leads to myocardial depression, a predisposition to ventricular arrhythmias, and characteristic ECG changes including right axis deviation, a widened QRS complex, and an R prime wave in lead aVR.

Secondary toxic effects include QTc prolongation mediated by potassium channel blockade, anticholinergic symptoms from M1 and M2 muscarinic acetylcholine antagonism, and hypotension due to peripheral alpha-1 adrenergic antagonism causing vasodilation.

Seizures are driven by GABA_A antagonism alongside the blockade of central nervous system sodium channels.

In asymptomatic overdoses, the standard of care dictates observing the patient for a minimum of 6 hours. Medical clearance requires the patient to be completely asymptomatic with completely normal vital signs and a normal ECG. Enhanced elimination via hemodialysis is ineffective at clearing TCAs. While multi-dose activated charcoal may reduce the TCA half-life, the high risk of aspiration associated with CNS depression and toxin-induced seizures generally outweighs the potential benefits.

The primary treatment for QRS widening (>100 msec) is sodium bicarbonate administered as 1-2 mEq/kg IV boluses every 3-5 minutes until the QRS narrows to <100 msec, targeting a serum pH between 7.45 and 7.55. (See Figure 2)

Bicarbonate boluses provide a large sodium load to overcome impaired myocyte conduction, and should be followed by a continuous infusion of 150 mEq sodium bicarbonate in 1L D5W at twice the maintenance rate.

Refractory hypotension should first be treated with isotonic crystalloid boluses up to 30 mL/kg, followed by norepinephrine as the preferred first-line vasopressor.

Toxin-induced seizures must be treated with benzodiazepines or other GABAa agonists such as propofol or phenobarbital; standard antiepileptics are entirely ineffective.

Patients exhibiting QTc prolongation require 2g of IV magnesium sulfate and close monitoring of magnesium and potassium levels, though Torsades de Pointes is relatively rare due to the protective tachycardia caused by anticholinergic effects.

For ventricular tachycardia or fibrillation that is refractory to defibrillation and multiple rounds of bicarbonate, a 1 mg/kg IV bolus of lidocaine is the preferred antidysrhythmic of last resort to displace the TCA from the sodium channels; this intervention must never be used for dysrhythmia prophylaxis.

Intralipid emulsion therapy (1.5 mL/kg bolus followed by a 0.25 mL/kg/minute infusion) is reserved as a final effort for refractory cardiovascular collapse, primarily in settings where V-A ECMO is unavailable.

Patients requiring aggressive intervention, such as intubation or continuous infusions, necessitate critical care monitoring and mechanical ventilation management. Following successful medical stabilization and extubation, a direct transfer and care handover to inpatient psychiatric services is required to address the underlying intentional ingestion.

Toxic effect	Treatment
Sinus rhythm with QRS >100msec	-Sodium bicarbonate, 1-2mEq/kg IV q3-5 minutes, goal reduction of QRS to <100msec/ pH 7.45-7.55 -Followed by sodium bicarbonate 150mEq in 1L D5W infusion at 2x maintenance rate target pH 7.4-7.55
Wide complex tachycardia or ventricular tachycardia	-Sodium bicarbonate, 1-2mEq/kg IV q3-5 minutes followed by sodium bicarbonate 150mEq in 1L D5W infusion at 2x maintenance rate target pH 7.4-7.55 Correct hypoxemia, acidemia, hypotension

	Lidocaine for refractory dysrhythmias despite sodium bicarb/defibrillation ; 1mg/kg IV bolus followed by infusion of 20-50mcg/kg/min Hypertonic saline (3%NaCl) 1-2mEq/kg bolus 2g IV magnesium sulfate over 2 minutes
Torsades de Pointes	DC cardioversion followed by Magnesium sulfate and/or Overdrive pacing
Hypotension	Isotonic crystalloid bolus up to 30mL/kg Sodium bicarb Norepinephrine in standard titrated doses
Seizures	Benzodiazepines Intubation with continuous sedation via propofol/ midazolam Consider phenobarbital load 10-20mg/kg for refractory seizures AEDs generally ineffective
Refractory poisoning	If other therapies fail and VA-ECMO unavailable, intralipid 1.5mL/kg bolus over 2 minutes followed by 0.25mL/kg/mn for 30-60 minutes

Figure 2: Effects of TCA and recommended treatment

Conclusion

TCA overdoses present a complex and highly lethal array of cardiovascular and neurological challenges. Rapid recognition of the toxidrome—specifically the pathognomonic ECG findings of a widened QRS and a terminal R wave in aVR—is paramount for initiating immediate, lifesaving sodium bicarbonate therapy. Although TCA utilization had previously declined, their recent resurgence in managing diverse chronic outpatient conditions dictates that acute care clinicians remain highly proficient in recognizing and reversing their toxicological effects.

Quiz questions:

- What ECG changes are characteristic of TCA toxicity?
 - QRS prolongation
 - QTc prolongation
 - R prime in aVR
 - All of the above**

Answer A correct because...

Answer B incorrect because.. etc

- What agent should be first line for seizures in the setting of a TCA overdose?
 - Levetiracetam
 - Phenytoin
 - Lorazepam**

d. Valproic acid

Answer A correct because...

Answer B incorrect because.. etc

3. True or false: hemodialysis is indicated for enhanced elimination in the setting of acute TCA overdose

a. True

b. False

Answer A correct because...

Answer B incorrect because.. etc