Cocaine and Sympathomimetics

Team Leaders

Khalid Aleedan & Aseel Badukhon

Done by

Noura Alhogail
Fouad Bahgat
Mohammed Alyousef
Amal Alshaibi
Khalid Aleisa

Revised by: Yara & Basel

Objectives

? Not found (':

Inshaallah Question won't be outside the slides (:

NOTES EXTRA BOOK IMPORTANT GOLDEN NOTES

Overview of Cocaine & Sympathomimetics

Perspective:

Amphetamine is the most common type in saudi arabia (rarely see cocaine), the newest type of sympathomimetic is flakka (click on it!)& it leads to severe aggression and abnormal behavior.

Cocaine, amphetamines, and derivatives of amphetamines are called sympathomimetics. These agents cause central nervous system (CNS) stimulation and a cascade of physiologic effects.

Clinical effects of Sympathomimetics

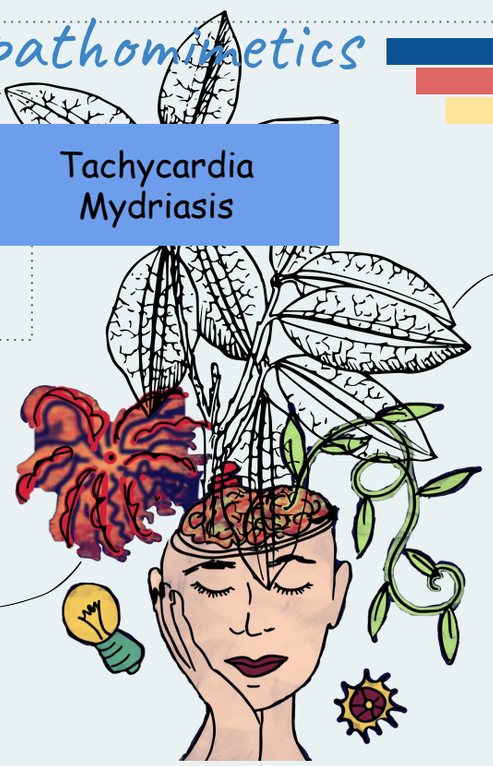
*Hypertension
*Hyperthermia

Tachycardia
Mydriasis

Diaphoresis
CNS excitation

لما تعصب يبقى شكلك عامل ايه (:

*These 2 in RED are the most dangerous because of their sequelae EX: HTN will if not managed properly pt can develop IC bleeding , Aortic dissection and flash pulmonary edema



Examples of Sympathomimetics:

Cocaine Amphetamines	Methylenedioxymethamphetamine Methamphetamine
Ephedrine and Ephedra Bath Salts	Khat and Methcathinone

*Flakka will reduce the re-uptake of the neurotransmitters. No effect on neurotransmitter release.
*Cocaine will increase release and reduce reuptake of neurotransmitters.
*Amphetamines will increase release in neurotransmitters. Does Not cause re-uptake of neurotransmitters.
All will work on sympathetic stimulation

Cocaine

Pathophysiology:

1 Adrenergic Stimulation

- Acute cocaine use causes release of dopamine, epinephrine, norepinephrine, and serotonin. most important effects are adrenergic stimulation by norepinephrine and epinephrine. **Norepinephrine** causes **vasoconstriction** by stimulating **alpha-adrenergic** receptors on vascular smooth muscle.
- **Epinephrine & Dopamine** increases **myocardial contractility** and **heart rate** through stimulation of **beta 1 adrenergic** receptors leading to an increase in cardiac output.

2 Catecholamines Reuptake Inhibition

- In addition to causing catecholamine release, the reuptake of these stimulatory neurotransmitters from synaptic clefts is inhibited, altering the normal balance between excitatory and inhibitory tone in the CNS.
- Cocaine increases sympathetic tone by increasing neurotransmitters in the synapse. (Image)

3 Anesthetic Agent

- Cocaine also is a local anesthetic agent, slowing nerve impulses from neuronal pain fibers by blocking the inward movement of sodium across cell membranes (phase 0 of the action potential).

4 Na Channel Blockade

- Sodium channel blockade across myocardial cells, similar to the class IA antidysrhythmic agents, is responsible for the occasional conduction abnormality with acute cocaine toxicity.

Sodium channel present in two sites, myocardial cells and nerve ending so the the blockade will lead to local anesthesia and arrhythmias.

-Sinus tachycardia is the MOST COMMON form of arrhythmia with cocaine and amphetamine
-Most dangerous form is wide complex tachycardia >> treat with IV SODIUM Bicarbonate. If still not managed, give lidocaine but the problem is it decreases seizure threshold, so give it after benzodiazepine.
-SVT (supraventricular Tachycardia) and Afib (Atrial fibrillation) are narrow complex vs wide complex tachycardia ex: torsades de pointes IMPORTANT DIFFERENCE there is pulse in the narrow complex vs No pulse in wide complex. We treat them with sodium bicarb before the antiarrhythmic drug.

Metabolism:

Cocaine metabolism occurs in

Liver

Primarily to the active metabolite **norcocaine**, which potentiates the parent drug.

Plasma

Ecgonine methyl ester (EME) via pseudocholinesterase (plasma cholinesterase). May be **protective** because it is a vasodilator.

Benzoyl ecgonine is a metabolite found in the plasma, produced primarily by non-ezymatic hydrolysis and is the metabolite identified by **urine** toxicology screens.

Methylecgonidine and its metabolite ecgonidine are products of cocaine pyrolysis (crack). Although it is less commonly assayed, **methylecgonidine also can be identified in the urine.**

The use of ethanol with cocaine may form **cocaethylene**, a metabolite that may potentiate the drug's stimulatory effects and lengthen the duration effect.

What substance do we check in urinary toxicology analysis to diagnose a pt with cocaine consumption?
Benzoylecgonine NOT COCAINE or EME

Genetic differences in the phenotypic expression of plasma cholinesterases may account for individual differences in susceptibility to cocaine toxicity.

Cocaine

Cocaine pharmacology & route of administration

Doctor went through it quickly (:

Route	Formula	Onset of action	Peak effect	Duration
Inhalation	Crack	8 sec	2-5 min	10-20 min
Intranasal	Cocaine HCL	2-5min	5-10 min	30 min
Intravenous	Cocaine HCL	Seconds	10-20 min	60-90 min
Oral	Cocaine HCL	30-60 min	60-90 min	Unknown
Skin Popping	Cocaine HCL	Unknown	Unknown	Unknown

Clinical Features:

Well, this is extremely important, half of the lecture is about it

Excitation of the sympathetic nervous system.

Moderate Toxicity	VS	Severe Toxicity
Alert and awake but may have diaphoresis,tachycardia, mydriasis, and hypertension **without organ damage**.		A more severely intoxicated patient may present agitated, combative (very agitated), and **hyperthermic.**

The most important difference between pt with Anticholinergic toxicity (dry) and cocaine or sympathomimetics is the diaphoresis.

Signs and symptoms of end-organ damage may be present,Emergencies.

- Patients may present with

Acute focal pain syndromes	Circulatory abnormalities	Delirium	Seizures

The clinical presentation depends on the dose, route of administration, and time to presentation after drug use.

Mortality is high with temperatures greater than **41.1°C**

Initial assessment and treatment should focus on rapidly fatal complications: Hyperthermia, hypertensive emergencies, cardiac dysrhythmias, Seizure

Cocaine

Fatal rapid complications of Cocaine Overdose

Complication	Description	Management
Hyperthermia	-Agitation with delirium increases the risk of hyperthermia. -Vasoconstriction and dehydration can compromise cooling, resulting in life-threatening hyperthermia with core temperatures exceeding 106 F (41.1 C). -Delay in recognition result in death. -Increased motor tone can release intramuscular (CK) with rhabdomyolysis and renal and electrolyte complications	-Rapid cooling. -Patients who sustain elevated core temperatures greater than (41 C) for more than **20 minutes** are likely to stabilize transiently, then develop fatal multisystem organ failure, often heralded by DIC. -Patients should have continuous monitoring of core temperature with a rectal probe. -It is crucial to reduce core temperature to (38.8? C) within **20 minutes**. -Cooling blankets are insufficient. Ice water, wet sheets with large fans, and packing the entire body in ice with continuous monitoring of core temperature can be used. -These patients often require aggressive **fluid resuscitation**. Water & Na depletion! pts with hyperthermia have higher mortality rate due to multi-organ failure (if it lasted more than 20 minutes), pulmonary hypertension, and rhabdomyolysis. Treat the pt rapidly, with cooling agents and benzodiazepines. The target temperature is 38.8
Hypertensive Emergencies	-Sequelae include : 1-Aortic dissection 2-Pulmonary edema 3-Myocardial ischemia and infarction 4-Intracranial hemorrhage 5-Strokes 6-Infarction of the anterior spinal artery. -Vasospasm can compromise perfusion to various organs. -Interstitial infarctions and mesenteric ischemia can occur, particularly in the body packers with large oral ingestion. Other local ischemic events include retinal vasospasm, renal infarction and placental insufficiency and infarction in the gravid uterus.	-Benzodiazepines restore the CNS inhibitory tone on the peripheral nervous system. -With evidence of end-organ damage, IV nitroglycerin or nitroprusside can be used. -Phentolamine, a direct alpha-adrenergic antagonist, is the antihypertensive of choice in Hypertensive Emergencies -It can be titrated slowly using repeat IV doses of 1 to 5 mg with blood pressure monitoring. **-Beta Blockers may cause paradoxical hypertension with cocaine.** **-Beta Blockers use in cocaine-related chest pain syndromes should be avoided.**
Dysrhythmias	-May not be noted until cardiac output abruptly diminishes, and the patient suddenly loses consciousness. -Sinus tachycardia is most common -Atrial fibrillation and other supraventricular tachycardias -Torsades de pointes or wide-complex tachycardias from **blockade of fast sodium channels on the myocardium** may deteriorate into poorly perfusing or fatal ventricular rhythms. -**Hyperkalemia** from rhabdomyolysis and myocardial ischemia can cause dysrhythmias.	-Atrial or ventricular. (Both types of dysrhythmias) -Atrial fibrillation and and supraventricular tachycardias are likely due to sympathetic stimulation and often respond to benzodiazepines. **Beta-adrenergic antagonists should be avoided.** -When the cause of a wide-complex tachycardia from cocaine is unknown, an empirical sodium bicarbonate, 1 to 2 mEq/kg IV bolus -Treats sodium channel blockade and potential cardiotoxicity from hyperkalemia. Lidocaine: -May increase seizure risk and mortality and is therefore reserved for patients with **ventricular dysrhythmias** for whom bicarbonate therapy has failed and who have **already received benzodiazepines** -Most useful for **ventricular dysrhythmias** with cocaine-associated MI. Amiodarone: -Is not well studied, but may be beneficial for ventricular dysrhythmias.

Cocaine

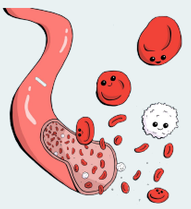
Other complications

Burns

oropharyngeal burns from the high temperature required to volatilize the drug

Crack Dancing

"crack dancing," a transient choreoathetoid movement disorder
(Can be treated by Benzodiazepines)



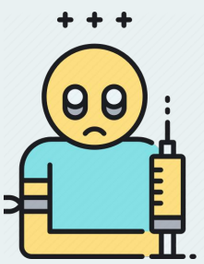
Abnormal Coagulation

DVT is reported with cocaine use, probably secondary to effects on coagulation.

Cocaine Washout

-People who binge with continuous use causes catecholamine depletion, dehydration, and poor nutrition.

-After the acute effects of cocaine have subsided, these patients with **"cocaine washout"** are profoundly sleepy but arousable and oriented, with normal vital signs or a mild sinus bradycardia..cocaine washout basically is due to the Depletion of catecholamine.. we don't do anything for the pt, he will be ok, just monitor his vital signs



Route of Administration related complications

Intranasal Cocaine use is associated with sinusitis and nasopalatine necrosis or perforation."When they Sniff the drug"

Transdermal injection of cocaine, or "skin popping," has similar types of complications.

Intravenous users have a high risk of infection with blood-borne viruses, local abscesses, and systemic bacterial infections, including botulism, and **endocarditis**.

Respiratory Complications

Pneumothorax, pneumopericardium, and pneumomediastinum occur from inhalational barotrauma. lung injury due to pushing air with high speed

Cocaine

Diagnostic Strategies:

Urine Analysis

-Urine drug screening is unlikely to change treatment because it measures a cocaine metabolite (Benzoylecgonine) that is typically present for 3 days after last use.

-Urine drug screening may be beneficial in:

1-To document possible abuse

2-To confirm cocaine as the unknown substance in body packers

3-To differentiate paranoia from drug-induced or psychiatric causes.

4-ECG

5-Sinus tachycardia ,wide complex tachycardia

-Urinalysis should be checked for myoglobin, which indicates rhabdomyolysis.

You should do Urine toxicology analysis for any patient you suspect drug abuse. We see Benzoylecgonine in a pt who took the drug through the past 3 days, our decision to treat the pt will be based mainly on the presentation s NOT on the results of the urine toxicology Analysis. We look at the analysis to differentiate between if the pt is having these presentations because of cocaine or severe psychosis or other medical illness.

ECG & Cardiac Markers

-Cyclic antidepressants and cocaine share class **Ia antiarrhythmic effects**.

-Accurate diagnosis of chest pain is problematic.

-ST segment elevation is confounded by the presence of early repolarization.

-Serial ECGs may be helpful.

-Creatine kinase (CK) serum CK-MB fraction, troponin I, and troponin T are more specific in patients with atherogenic coronary disease.

Neurological Investigation

Severe, persistent headache despite normalization of blood pressure may occur with a SAH (subarachnoid hemorrhage) and warrants head CT and, if the scan is negative, lumbar puncture.

Observe until pt is pain free, if the ecg is normal with cardiac markers, it is safe to discharge the pt.

Initial Evaluation of Patients with Sympathetic Stimulation:

1-Rapid assessment of vital signs,especially core temperature

2-Rule out hypoxia ,hypoglycemia

3-Pharmacologic sedation with benzodiazepines

4-Electrocardiogram

5-Urinalysis

6-Serum creatinine phosphokinase

Cocaine

Differential Considerations: Important!

- Sedative-hypnotic withdrawal
- Amphetamines and its derivatives you can't differentiate it clinically only in urine toxicology analysis
- Heatstroke. very high temp
- Infection should be considered in all hyperthermic patients.

Differential Diagnosis of Agitated Delirium:

Metabolic causes	Electrolyte abnormalities	Hypoglycemia, Hypoxia	Urima\hyperammonemia
Structural lesions of the CNS	Trauma, stroke	Hemorrhage	Mass
Endocrine Diseases	Thyrotoxicosis		
Infections	Bacterial\Viral meningitis	Bacterial\Viral encephalitis	
Toxicologic causes	Sympathomimetics\ Stimulants	Cocaine, Amphetamines and derivatives	Caffeine, Phencyclidine\Ketamine
	Anticholinergics	Serotonin Syndrome	Sedative-hypnotic withdrawal
Other	Heatstroke	Postictal state	

Cocaine

Management:

- After initial **airway assessment** 1st thing note:do intubation early if u suspect intracerebral hemorrhage before sending him to CT , 2nd most imp thing is the ECG
- Physical restraints to obtain complete vital signs and to secure IV access.
  Remember, they are agitated (:
- Empirical therapy with IV dextrose and thiamine or assessment with a bedside blood glucose monitor.
- IV benzodiazepines may be necessary.
- Most cocaine toxic patients have salt and water depletion and require vigorous IV crystalloid replacement

Pharmacologic Sedation

- In adults, IV diazepam can be administered in increments of 10 mg every 5 minutes until sedation is achieved.
- In wildly agitated patients in whom 20 to 30 mg of diazepam has no notable effect,the increments may be increased carefully by 20 mg each subsequent dose with close monitoring.
- If additional doses of Lorazepam or Diazepam are given then close monitoring of respiratory status is necessary.

- Because cocaine agitation can be indistinguishable or coexist with alcohol intoxication, the presence of alcohol can have synergistic depressant effects on the respiratory centers.
- Titration of Benzodiazepine is important in all patients; you'll observe the effects of one dose before you start the other
- **Butyrophenone** (antipsychotic agent) is used in pts with normal vital signs; lacks respiratory depression effect but can cause dysrhythmias.

Management of Stimulant-Induced Hyperthermia:

-Early identification of elevated core temperature
-Large-bore intravenous access with rapid infusion of crystalloid
-Sedation and muscle relaxation with benzodiazepines
-Rapid cooling within 20 min*

-Urinalysis for myoglobinuria
-Paralysis and intubation if necessary

-Foley catheterization to monitor output
-Laboratory analysis for organ function
1-Serum chemistries/creatinine /CK
2-Liver function
3-PT/PTT/fibrin split products
4-Bacterial cultures**

*Ideally with ice water immersion.
**Consider lumbar puncture or antibiotic therapy, especially in injection drug users.

CK:creatine kinase, PT:prothrombin time ,PTT:partial thromboplastin time

Cocaine

Cocaine related chest pain

-Cocaine induces coronary vasoconstriction and increases myocardial oxygen demand.
-Platelet aggregation is enhanced through prothrombogenic and antifibrinolytic pathways.

Causes of Cocaine related chest pain

The causes of cocaine-related chest pain are diverse, including aspirated foreign bodies or pneumothorax or pneumo-mediastinum from inhalational barotrauma.

Cardiac chest pain	Noncardiac chest pain
-Endocarditis -Pericarditis -Ischemia/infarction -During acute intoxication -After acute intoxication -Coronary stent thrombosis	-Pneumothorax -Pneumomediastinum -Pneumopericardium -Aortic dissection -Pulmonary infarction -Infection -Foreign body aspiration

-Total CK in urine: come from both heart and striated muscle so we do Ck mb fraction its for myocardial only it should be only 10% of the total , if more than this heart injury.
-ECG showing St elevation all chest & limb leads, upward "smiling". In MI, it's either flat or convex upward elevation.

Chest radiograph is used to identify:
-Aspirated foreign bodies
-Pneumothorax or pneumomediastinum from inhalational barotrauma.

Management

-Benzodiazepines decrease myocardial oxygen demand by limiting peripheral stimulation and should be given early.
-Aspirin and nitrates also should be administered.
-In patients meeting ECG criteria for MI with persistent chest pain and hypertension and a clear history of acute cocaine intoxication > coronary vasodilation with IV phentolamine (1 mg) given slowly should be considered.
-Morphine sulfate also can be used to treat pain.
-Patients with persistent chest pain and ST segments strongly suggestive of MI can be considered for percutaneous intervention in the catheterization laboratory or thrombolytic therapy, assuming there are no contraindications such as uncontrolled severe hypertension. Don't give fibrinolytic unless u reduce systolic blood pressure. IF BP is ABOVE 180, FIBRINOLYTIC therapy is CONTRAINDICATED.
-Beta-adrenergic antagonists (Beta blockers), including labetalol, are **contraindicated** during acute cocaine toxicity. Give phentolamine

Risk group

-Fever and shortness of breath should prompt consideration of pneumonia, pulmonary infarction, or endocarditis with septic pulmonary emboli in IV drug abuse.
-Patients with positive serum enzymes for MI have significant angiographic stenosis
-Patients without positive serum enzymes,18% still have significant disease by angiogram.
-Other predictors of significant disease in this group included elevated cholesterol and prior diagnosis of coronary disease or MI.
-Patients with a history of coronary stent placement are at a high risk of thrombosis with cocaine use.

Cocaine

Cocaine Body Packers (important)

-Before crossing international borders, "body packers" ingest cocaine that has been wrapped tightly into condoms or other latex products and sometimes coated in wax.
-Each packet can contain approximately 10 g of cocaine, and packers may swallow as many as 150 packets. lethal dose 30 mg can kill you, if ur addicted then lethal dose can be inc. to 1.1 g of cocaine.
-Body packers are likely to know the exact number of packets they ingested.

Body Stuffers (important)

-A "body stuffer" is an individual who attempts to conceal evidence of cocaine possession by swallowing the drug while pursued by law enforcement officials.
-These are usually unplanned events with generally small quantities of drug intended for personal use.

Basically, when the abusers are sitting together in the استراحة to start taking the drugs and the police يطب عليهم and then they swallow all the drug they have immediately عشان لا يتمسكوا و يروحوا عند خلاتهم XD

- A body packer may present without symptoms to the ED.
- The body packer should be placed immediately on continuous cardiac monitoring, with large bore IV access.
- An abdominal radiograph (EX:x-ray) may confirm foreign bodies.
- When uncertainty persists, a contrast study is warranted.

Diagnosis & Management

-When evidence of cocaine toxicity is manifest, rapid transportation to the **operating room** may be the only way to save these patients.
-Benzodiazepines, neuromuscular blockade, or sodium bicarbonate administration may be required en route.
-CT and contrast abdominal radiographs may fail to detect potentially fatal quantities of cocaine.
-Endoscopic retrieval is discouraged because of concern over packet rupture during the procedure.

Speedballing (important)

Patients who are "**speedballing**," using IV heroin and cocaine together, may be initially **sedated**, and administration of naloxone may reveal the underlying cocaine intoxication by stopping the heroin effect and that will cause a sudden agitation. So we need to make sure there's an enough amount of benzodiazepines. Speedballing: a professional abuser, he knows that these drugs will make him so agitated and it may make him lose control, so he takes with it some other benzo, morphine, heroin to make a balance ويعمل دماغ مضبوطة (؛

You should know ALL these TERMS like Speedballing, body packers and body stuffer very well

Cocaine

Disposition:

Discharge	ICU	ECG Changes
-Can be discharged after the acute intoxication resolves. -These patients may be extremely sleepy from catecholamine depletion, and it is best to discharge them with a responsible adult. -After a 12-hour monitored observation period, patients with a benign clinical course and negative serum enzyme markers can be discharged.	-Patients who develop complications should be admitted to the intensive care unit for further treatment.	-Patients with chest pain who are acutely intoxicated and who show dynamic changes on the ECG, dysrhythmias, or congestive heart failure or patients requiring vasodilators or reperfusion should be admitted. -These patients require further evaluation of the extent of preexisting reversible ischemia and intervention to encourage cessation of drug use.

Admission Criteria For Cocaine Related chest pain			
	-Persistent chest pain -Electrocardiogram changes -Dysrhythmias or conduction abnormalities	-CHF/cardiogenic shock -Elevated enzymes -Requiring vasodilation	-Preexisting CAD or a stent placement -Multiple risk factors for CAD -CAD coronary artery disease , CHF congestive heart failure

Body Packers Disposition

- Body packers need to be observed until all packets have passed.
- Ideally, these patients have had three packetfree stools, a reliable packet count consistent with the ingestion, and a negative contrast radiographic study.

Despite managing the HTN the pt has persistent headache YOU SHOULD DO CT ! but subarachnoid hemorrhage can be missed in CT so after it if still headache do LP looking for Xanthochromia

Other Stimulants

Amphetamines

More common in KSA than cocaine

-Enhance release of catecholamines from presynaptic nerve terminals
-Does not affect the reuptake like cocaine
-Usually taken as pills, but occasionally are crushed and injected.

-CNS stimulation results in nearly identical sympathomimetic effects to those from cocaine,but not with the same frequency or intensity.
-Amphetamines Patients are at risk for: Hyperthermia ,hypertensive emergencies, Dysrhythmias, myocardial ischemia, hyperkalemia associated with rhabdomyolysis.

-**Don't block sodium channels** and only **minimally** affect presynaptic reuptake of catecholamines.
-Although urine drug screens can identify amphetamines, they are of little utility in treating an intoxicated patient.
-The management follows the same guidelines as for cocaine, although the **duration of toxicity tends to be longer for amphetamines**.

Methylenedioxymethamphetamine

(MDMA—"Ecstasy," XTC, Adam)

is a chemically **modified amphetamine** (longer effect) originally taken orally at all-night dance parties, or "raves." Patients describe the euphoria allowing "closeness to others," so it is sometimes called the "love drug."

-**Life-threatening hyponatremia**, may alter release of endogenous stores of **vasopressin**.
-Urine samples with a relatively high urine sodium level, similar to SIADH.
-Unless seizures or other neurologic events are present, patients can be treated supportively with fluid restriction.
-Normal saline or other crystalloids may worsen the hyponatremia because these patients are likely to **retain more free water than sodium**.

due to SIADH

-Their fluid intake should be restricted unless severe hypovolemia exists, and they should be treated with hypertonic saline for neurologic impairment.
-In contrast to other amphetamines, chronic MDMA use causes potentially irreversible neurologic damage to serotonergic neurons.

Methamphetamine

Methamphetamine Methamphetamine, known as "crank" and "crystal meth," is a fat-soluble, smokable, designer amphetamine.

Complications from methamphetamine use are similar to those from other sympathomimetics.

The duration of action can be significantly longer, however, with some paranoid delusions persisting for 15 hours. There is No Na blocking therefore no wide complex tachycardia with amphetamine like cocaine

Key Concepts:

-Rapid sedation with an IV benzodiazepine is the key for most symptoms from cocaine and other stimulants.
-Hyperthermia is a high-risk sign, and body temperature must be reduced rapidly.

-Beta adrenergic blockade may cause paradoxical hypertension and increase coronary vasoconstriction and is generally contraindicated
-Wide complex rhythms secondary to cocaine may respond to IV bicarbonate therapy

-Cocaine body packers who become symptomatic need **immediate surgery**
-Amphetamine symptoms and effects **last longer** than those produced by cocaine

-If pt was agitated, do physical restraint not moer 20 min to establish iv line
-41.1 can kill the pt 38.8 goal don't forget rectal probe for monitoring
-ANY pt with any drug overdose due to attempting suicide should be referred to psychiatry

IMPORTANT ! Secure the airway
If unconscious and cannot deal with secretions incubate the patient

Summary

Clinical Effects of Sympathomimetics:

These agents cause central nervous system (CNS) stimulation and some the important clinical effects are:

Hypertension[1] | **Hyperthermia[2]** | **Diaphoresis[3]**

1-Hypertension can lead to life threatening conditions such as stroke and aortic dissection
2-Mortality is high with core temperature of 41.1; hypertension and hyperthermia are among the most important complications of stimulants overdose and need an extensive care and management
3-Major difference between anticholinergic toxidrome and Sympathomimetics

Cocaine

Pathophysiology and metabolism	Clinical Features and complications	Diagnosis and Evaluation	Management and Disposition
-Release of dopamine, epinephrine,norepinephrine, and serotonin -Inhibit reuptake of these stimulatory neurotransmitters -Local anesthetic agent (blocks the inward movement of sodium, similar to the class IA drugs) **Metabolism:** 1-Liver → norcocaine (potentiates cocaine) 2-Plasma → ecgonine methyl ester via pseudocholinesterase	-Naloxone may reveal cocaine intoxication in **Speedballing** (using IV heroin and cocaine). ->41.1°C →Mortality is high. **Complications:** -Hyperthermia -Hypertensive emergency -Cardiac dysrhythmias -Others such as Cocaine washout **Cocaine related chest pain causes:** Noncardiac, Foreign body aspiration, Cardiac, or due to Coronary stent thrombosis	-Urine drug screening measures the metabolite (benzoylecgonine). -Accurate diagnosis of chest pain -ECG -Cardiac enzymes -Urinalysis -Assessment of vital signs -Serum creatinine phosphokinase -Rule out hypoglycemia and hypoxia -Sedation with benzodiazepines	**Management:** -Airway assessment -Physical restraints -IV dextrose and thiamine -IV benzodiazepines (diazepam) -Hyperthermia: rapid cooling + rectal probe monitoring -Phentolamine in hypertensive emergencies (NOT B-blockers) -Wide-complex tachycardia → IV $NaHCO_3$ **Disposition:** -Discharged after acute intoxication resolves -Complications → ICU -Intoxicated with chest pain and cardiac manifestations → admitted -After 12-hour observation: benign clinical course + negative markers → discharged -Body packers observed until all packets pass

Amphetamines

-Enhance release of catecholamines
-Effects nearly identical to Cocaine
-Do not block sodium channels
-Same management as cocaine, but with longer duration of toxicity

Methylenedioxyamphetamine (Ecstasy)

-Life-threatening hyponatremia
-Alter release of vasopressin, high urine sodium similar to SIADH
-Treated by fluid restriction,if Hypovolemic —> hypertonic saline for Neurologic impairment
-Irreversible damage to serotonergic neurons

Methamphetamine (crank)

-Fat-soluble, smokable amphetamine
-Paranoid delusions (15 hrs)
-Longer duration of action
-There is no Na channel blockade so wide complex tachycardia

Body Packer VS Body Stuffer

-Ingest cocaine wrapped in latex.
→ On continuous cardiac monitoring.
-Evidence of cocaine toxicity → rapid transportation to the operating room.
-Large quantities of drugs

-Conceal evidence of cocaine possession by swallowing
-Small quantity of drugs

Hey there! Have fun studying toxicology (;

How toxic is your knowledge

(Doctor's slides)

1)21 years old male brought to you in emergency department agitated sweating,BP 205/110 Pulse 140/min temperature is 39 c The most likely diagnosis is
A. Iron overdose
B. Amphetamine overdose
C. Antihistamine overdose
D. ASA overdose

2) Which one of the examples is a stimulant drug ?
A. Barbiturate
B. Marijuana
C. Morphine
D. Bath Salts

3) Cocaine metabolized in which of the following ?
A. Lung .
B. Kidney .
C. Pancreas .
D. Liver & Plasma .

4) 20 year old patient present to you with sweating, agitation, tachycardia and mydriasis . This presentation can be caused by which of the following ?
A. Depressant drugs .
B. opioid .
C. cannabis.
D. Amphetamine .

5) 30 years old patient come to you after ingestion of large amount of cocaine with tachycardia and hypertension .you perform ECG which shows wide complex tachycardia . which of the following will be your first line medication to be given ?
A. Sodium bicarbonate .
B. Amiodarone .
C. procainamide .
D. lidocaine .

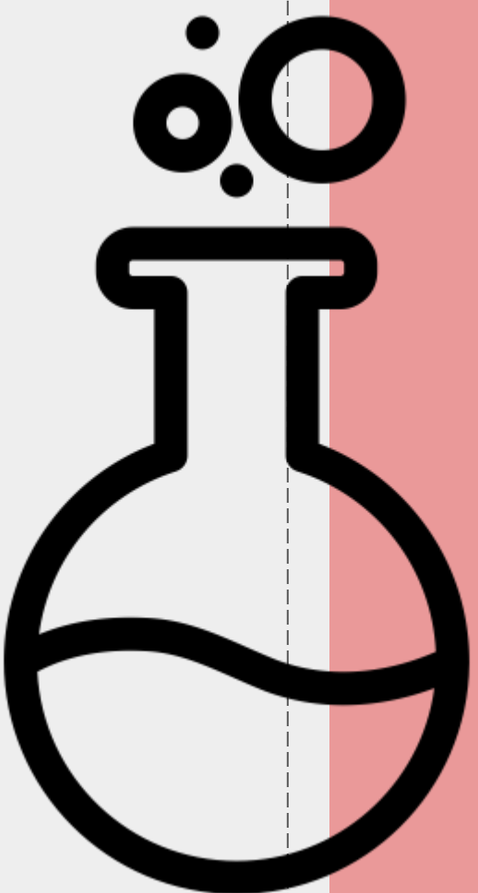
6) Which one of the following is a fatal complication of stimulant toxicity ?
A. Hyperthermia
B. Hypothermia
C. Hypokalemia
D. Bradycardia

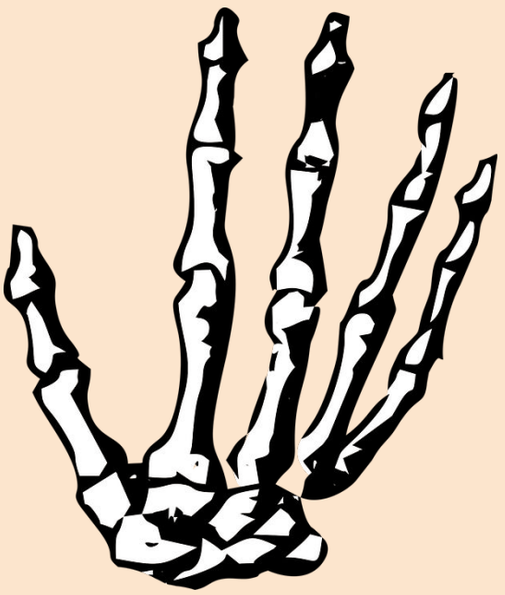
7) You are treating a young patient with cocaine overdose, which of the following drugs you should avoid while treating such patients?
A- Benzodiazepines
B- Beta blockers
C- Alpha blockers
D- Sodium bicarbonate

8) 29-year-old male is brought to the emergency department after using amphetamine in a party. Which of the following complications may occur in this patient?
a. Convulsions
b. Hypothermia
c. Hypotension
d. Bradycardia

1-B
2-D
3-D
4-D
5-A
6-A
7-B
8-A

Thank you and good luck!





Very Toxic but you are gonna do it!

A+ is yours (:

- Email us at:

436toxicology@gmail.com

How well do you think we have done? We are waiting for your feedback!

Click here!

- Theme was designed by: Aseel Badukhon
- Logo was designed by: Norah Alhogail