

Substance use disorder

Toqa rakhameen Toqa al-qatatsheh

Definition and diagnostic criteria

Definition: A chronic condition in which an uncontrolled pattern of substance use leads to significant physical psychological, and social impairment or distress, with continued use despite substance-related problems.

Types of Drugs:

Stimulants (Uppers)	Depressants (Downers)	Hallucinogens
What do they do?	What do they do?	What do they do?
Speed up the brain and central nervous system.	Slow down the brain and central nervous system.	These drugs alter the user's state of consciousness. (Distort auditory and visual sensations)
Examples: Caffeine (coffee, energy drinks, tea) Nicotine (cigarettes) Amphetamines (meth, ecstasy) Speed "Bath salts" Cocaine and Crack Cocaine	Examples: Alcohol (beer, wine, vodka, tequila, gin, etc.) Heroin Tranquilizers Sleeping Pills Marijuana	Examples: LSD Ecstasy Magic mushrooms Peyote PCP

- Diagnostic criteria:
- At least two of the following within a 12-month period:
- Using substance more than originally intended.
- Persistent desire or unsuccessful efforts to cut down on use.
- Significant time spent in obtaining, using, or recovering from substance.
- Craving to use substance.
- Failure to fulfill obligations at work, school, or home.
- Continued use despite social or interpersonal problems due to the substance use.
- Limiting social, occupational, or recreational activities because of substance use.
- Use in dangerous situations (e.g., driving a car).
- Continued use despite subsequent physical or psychological problem (e.g., drinking alcohol despite worsening liver problems).

Epidemiology

- Sex: ♂>♀
- Alcohol and nicotine use are most common.
- InJordan: Nicotine is the MCtype.
- One-year prevalence of any substance use disorder in the United States is approximately 8%.

Psychiatric symptoms

- Mood symptoms are common among persons with substance use disorders.
- Psychotic symptoms may occur with some substances.
- Personality disorders and psychiatric comorbidities (e.g., maj or depression, an xiety disorders) are common among persons with substance use disorders.
- It is often challenging to decide whether psychiatric symptoms are primary or substance-induced.

Risk factors

• Environmental:

Qusing the substance at an early age, or experiencing physical, sexual, or emotional abuse.

- Social and legal policies
- Acceptability
- Genetic predisposition:

These include a family history of substance use disorder or

having a mental health disorder like depression, ADHD

- Peer factors :
- Substance uses during birthdays or parties
- O Excessive time spending with substance using peers



Tobacco related disorders

• Substance: Nicotine from the tobacco plant (consumed in cigarettes, cigars, pipes, e-cigarettes) Clinical features:

Mechanism of action :

 stimulates nicotinic receptors in autonomic ganglia → sympathetic and parasympathetic stimulation

Epidemiology:

- Approx. 13% of adults in the US smoke cigarettes.
- Most prevalent cause of preventable morbidity and mortality in the US.

Assessment:

• Smoking history is measured in pack years, which is used to quantify a person's lifetime exposure to tobacco.

Tobacco intoxication

Euphoria

Tachycardia, mild HTN, weight loss

Restlessness, anxiety

Increased gastrointestinal motility

Insomnia

Tobacco Withdrawal

Dysphoria, depressed mood.

Irritability, frustration, anger, restlessness, anxiety.

Insomnia

Impaired concentration

Increased appetite, weight gain

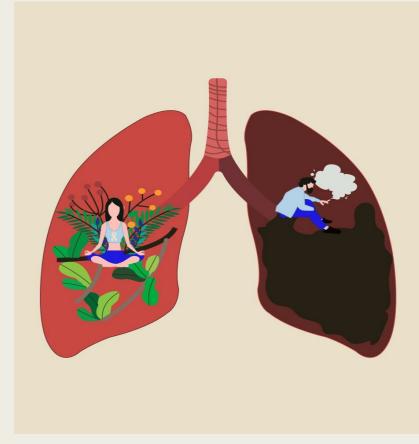
Treatment:

- Counseling and support
- Varenicline (partial nicotine receptor agonist): reduces positive symptoms and prevents withdrawal.
- Bupropion: reduces craving and withdrawal symptoms

• Nicotine replacement therapy (inhaler, lozenges, transdermal patch, nasal spray, gum).

Complications:

- COPD
- Cardiovascular disease
- Cancer (e.g., lung cancer)



Alcohol

- alcohol is a potent CNS depressant. activates gamma-aminobutyric acid (GABA), dopamine, and serotonin receptors in the central nervous system (CNS)
- Thus, alcohol abuse can produce serious temporary psychological symptoms including depression, anxiety, and psychoses.
- On Long-term, escalating levels of alcohol consumption can produce tolerance as well as adaptation of the body that cessation of use can precipitate a withdrawal syndrome.
- Lifetime prevalence of alcohol use disorder in the United States is 5% of women and 12% of men.



Alcohol intoxication

- Alcohol intoxication (also called simple drunkenness) according to DSM-5 diagnostic criteria are based on evidence of recent ingestion of ethanol, maladaptive behavior, and at least one of several possible physiological correlates of intoxication.
- Signs of Alcohol Intoxication
- 1. Slurred speech
- 2. Dizziness
- 3. incoordination
- 4. Unsteady gait
- 5. Nystagmus
- 6. Impairment in attention or memory
- 7. Stupor or coma
- 8. Double vision

Treatment:

- Perform an ABCDE survey to assess hemodynamic and respiratory stability
- Check vital signs and glucose
- Administer naloxone for opioid overdose if co-ingestion is suspected.
- Provide supportive care when appropriate
- Manage dehydration and hypovolemia.
- Respiratory support.
- Prophylactic dose of thiamine supplementation (for Wernicke's).
- Electrolytes repletion .
- •Gastrointestinal evacuation (e.g., gastric lavage, induction of emesis, and charcoal) is not indicated in the treatment of EtOH overdose, except with significant amounts within 30-60 min
- A CT scan of the head may be necessary to rule out subdural hematoma or other brain injury.
- •Severely intoxicated patients may require mechanical ventilation with attention to acid—base balance, temperature, and electrolytes while they are recovering

Complications of chronic alcohol use

Wernicke's encephalopathy

An acute, reversible condition caused by severe thiamine (vit B1) deficiency, often due to chronic heavy alcohol use. The classical triad (seen in about a third of patients):

1. Confusion

2.Oculomotor dysfunction (vertical nystagmus is the most common), diplopia .

3. Gait ataxia

Other manifestations:

- Autonomic dysfunction: hypotension, syncope, hypothermia.
- peripheral neuropathy: paresthesia, foot drop, decreased DTR.
- Cardiovascular dysfunction: tachycardia, exertional dyspnea.



Treatment:

- immediate IV
 administration of high dose Vit B1
 - Thiamine must be administered before IV glucose infusion

Complications of chronic alcohol use

Korsakoff syndrome:

- Chronic thiamine deficiency, especially in patients with alcohol use disorder, frequently progresses to Korsakoff syndrome, which is characterized by :
- 1. Irreversible personality changes, apathy, indifference.
- 2. Anterograde and retrograde amnesia, (anterograde is more common than retrograde).
- 3. Confabulation, Patients create fabricated memories to fill in the gaps of their memory.
- Other manifestations :
- Disorientation to time, place, and person.
- Hallucination

Treatment:

- -Oral thiamine supplementation to prevent further progression to irreversible complications.
- -Psychiatric and psychological therapy.
- Memory strengthening exercises and aids

Alcohol withdrawal Clinical Presentation

Bigns and symptoms include insomnia, anxiety, hand tremor, irritability, anorexia, nausea, vomiting, autonomic hyperactivity (diaphoresis, tachycardia, hypertension), psychomotor agitation, fever, seizures, hallucinations, and delirium tremens (DTs)

TABLE 7-3. Alcohol Withdrawal Symptoms

Alcohol withdrawal symptoms usually begin in 6–24 hours after the last drink and may last 2–7 days.

Mild: Irritability, tremor, insomnia.

Moderate: Diaphoresis, hypertension, tachycardia, fever, disorientation.

Severe: Tonic-clonic seizures, DTs, hallucinations.

Delirium tremens

It is the most severe form of ethanol withdrawal, and it is a medical emergency that can result in significant morbidity and mortality.

Occurs in 5% of patients

Clinical features:

- Altered mental status (confusion)
- Autonomic hyperactivity (such as tachycardia, diaphoresis, fever, anxiety, insomnia, and hypertension)
- Perceptual distortions, most frequently visual or tactile hallucinations, and fluctuating levels of psychomotor activity, ranging from hyper excitability to lethargy.

Onset: usually 72–96 hours after cessation of or reduction in alcohol consumption

- Patients with delirium are a danger to themselves and to others, Because of the unpredictability of their behavior, patients may be assaultive or suicidal or may have hallucinations or delusional thoughts.
- Untreated DTs has a mortality rate of 20%

Treatment of alcohol withdrawal

- Minor alcohol withdrawal syndrome may not need pharmacotherapy in all cases. The patient needs supportive care in a calm and quiet environment and low lighting and observation for a period of up to 36 h, after which he is unlikely to develop withdrawal
- Benzodiazepines are the mainstay of management of alcohol withdrawal states, followed by anticonvulsants, which may be considered in mild withdrawal states due to their advantages of lower sedation and lower chances of dependence.
- Moderate to severe alcohol withdrawal syndrome Without seizures or DT, patients should be started immediately pharmacological therapy, while monitoring the clinical signs of tachycardia and hypertension

Treatment of alcohol withdrawal Cont.

- Severe alcohol withdrawal with seizures, the occurrence of seizures during the alcohol withdrawal period is indicative of severe alcohol withdrawal. Seizure prophylaxis with lorazepam intravenously must be given to all patients with seizures in the current withdrawal period at presentation
- Severe alcohol withdrawal with DT, the Treatment is by achieving a calm, but awake state ordefined as a sleep from which the patient is easily aroused. done by using intravenous diazepam while closely monitoring the patient during the procedure. Refractory DT can be managed with phehobarbital or adjuvant antipsychotics.
 - Detoxification is the process of weaning a person from a psychoactive substance by gradually tapering the substance or by substituting it with a cross-tolerant pharmacological agent and tapering it.
 - Rehabilitation for alcoholism is recommended.

Treatment of alcohol withdrawal

First-line treatment

Naltrexone:

Opioid receptor antagonist

Reduces desire and the "high" associated with alcohol.

Will precipitate withdrawal in patients with physical opioid dependence.

Acamprosate:

Thought to modulate glutamate transmission.

Use for relapse prevention in patients who have stopped drinking (postdetoxification).

Major advantage is that it can be used in patients with liver disease.

Contraindicated in severe renal disease.

Side effect as diarrhea

Second-line treatment

Disulfiram:

Blocks the enzyme aldehyde dehydrogenase in the liver, causing aversive reactions to alcohol (flushing, headache, nausea/vomiting, palpitations, shortness of breath) due to catecholamine release

Absolute Contraindication with severe cardiac disease, pregnancy, and psychosis.

Liver function should be monitored.

Best used in highly motivated patients, as medication adherence is an issue.

Topiramate:

Anticonvulsant that potentiates GABA and inhibits glutamate receptors.

Reduces desire for alcohol and decreases alcohol use.

Sedatives: Benzodiazepines



Diazepam, oxazepam, lorazepam

•Medical uses (anxiety, alcohol and barbiturates withdrawal)

Classic overdose presentation:

- ONS depression with normal vitals
- Altered mental status
- Slurred speech
- Ataxia
- Rarely cause respiratory depression (safer drugs)

In case of overdose: use flumazenil

- Antagonist of benzodiazepine receptor.
- -FDA-approved clinical uses for flumazenil include reversal agents for benzodiazepine overdose.
- Overdose has low mortality rate.
- -Flumazenil may cause withdrawal seizures in patients with a history of seizures

Benzodiazepine withdrawal

- -Occurs with abrupt cessation in chronic user.
- Timing depends on drug
 Long-acting BZD → longer washout

Presentation:

- Tremors
- Anxiety
- Depressed mood ("dysphoria")
- Hypersensitivity to sensations (noise, touch)
- Psychosis
- Seizures

Sedatives: Barbiturates



Phenobarbital, pentobarbital

- •Potentiate the effects of GABA by binding to the receptor and increasing duration of chloride channel opening.
- •Used in the treatment of epilepsy and as anesthetics
- •have a lower margin of safety relative to BZDs.
- They are Coacting in combination with BZDs, respiratory depression can occur.
- they are physiologically addictive if taken in high doses over 1 month
- Overdose: respiratory depression

• Abrupt abstinence after chronic use can be life threatening.

Clinical Presentation:

- •Signs and symptoms of withdrawal are the same as these of alcohol withdrawal.
- •Tonic-clonic seizures may occur and can be life threatening.

Treatment:

•Benzodiazepines (stabilize patient, then taper gradually).





- Opioid overdose results from the toxic effects of exogenous opioid
- •Deaths related to opioid overdose have been steadily increasing in the United States over the past two decades because of a sharp increase in the prescription of opioid for chronic pain and increasing amounts of illegally manufactured fentanyl.

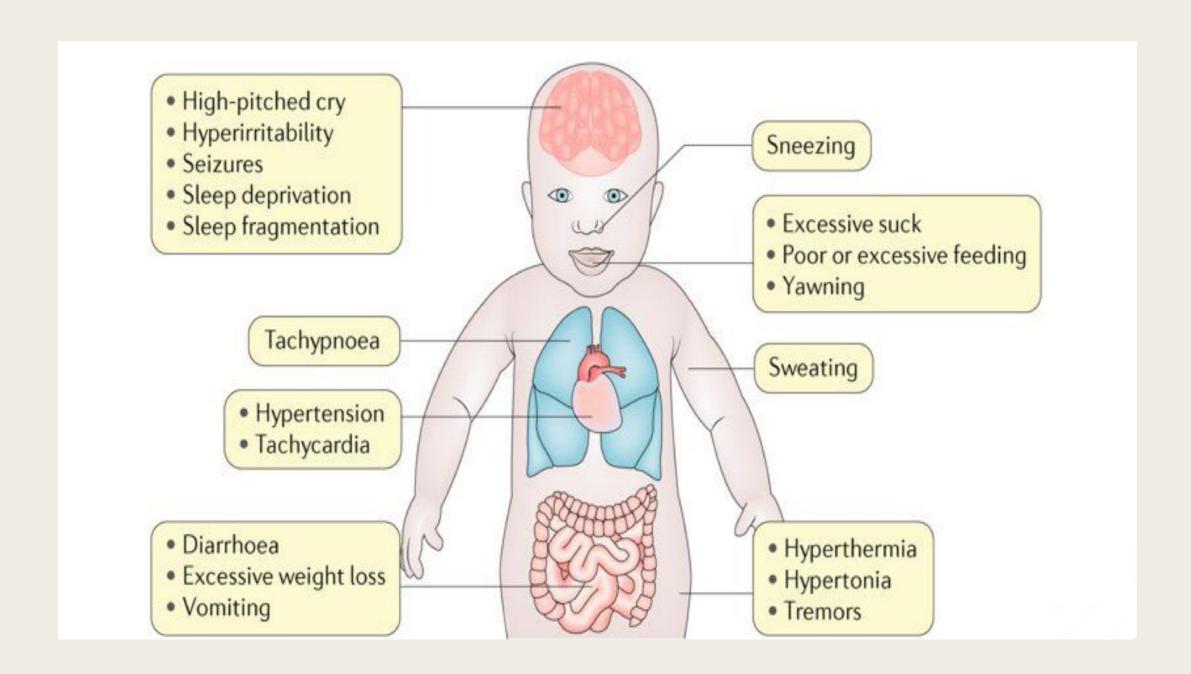
Common clinical features of opioid overdose include:

- Respiratory depression
- CNS depression
- miosis.

Treatment of suspected opioid overdose requires:

- •Airway management and prompt assessment of the need for naloxone to counter opioid induced respiratory depression, which can be **fatal**. Inpatient admission is indicated for patients with ongoing respiratory depression, overdose from long-acting opioid, or medical complications from an opioid overdose.
- •All patients with a non iatrogenic opioid overdose should undergo an assessment for substance use disorder and be discharged with take-home intranasal naloxone.

Opioid withdrawal in infants



Stimulants: Amphetamine

- •Potent stimulant by increasing synaptic levels of the biogenic amines, dopamine, norepinephrine and serotonin.
- •Amphetamine are FDA-approved for treatment of attention deficit-hyperactivity disorder (ADHD) and narcolepsy.

Symptoms of amphetamine intoxication include:

- Euphoria
- Dilated pupils
- Tachycardia
- Chest Pain
- Amphetamine withdrawal can cause prolonged depression.
- Complications of their long half-life can cause: ongoing psychosis, even during abstinence,
- so treatment is: sedation and observation with antipsychotics .

Stimulants: Cocaine

- •Cocaine blocks the reuptake of dopamine, epinephrine, and norepinephrine from the synaptic **cleft**, causing its stimulant effect
- Overdose can cause:
- O Death secondary to cardiac arrhythmia
- \bigcirc MI
- O Seizure
- Respiratory depression.
- Treatment of cocaine use disorder:
- There is no (FDA)-approved pharmacotherapy for cocaine use disorder.
- Off-label medications are sometimes used as naltrexone
- Psychological interventions are the mainstay of treatment.

Cocaine withdrawal

- Abrupt abstinence is not life threatening.
- causes post-intoxication depression .
- Occasionally, these patients can become suicidal.
- ●With mild-to-moderate cocaine use, withdrawal symptoms resolve within 72 hours with heavy, chronic use, they may last for 1–2 weeks.
- •Treatment is supportive, but severe psychiatric symptoms may warrant hospitalization.

Thank you