OPIUM

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- Opium poppy, *Papaver somniferum var. album*, is the species of plant from which opium and poppy seeds are extracted.
- The Latin botanical name means, loosely, the "sleep-bringing poppy, white form", referring to the sedative properties of some of these opiates

- The plant itself is also valuable for ornamental purposes, and has been known as the "common garden poppy",
- Poppy seeds of *Papaver somniferum* are an important food item and the source of poppyseed oil, a healthy edible oil that has many uses. Poppy seeds are called Khuskhus in Hindi, Gasagasalu in Telugu, Gasagasa in Kannada, Posto dana in Bengali.

- The opium poppy is the principal source of all natural opiates.
- Opium (raw opium) is the latex harvested by making incisions on the green capsules (seed pods). Poppy straw is the dried mature plant except the seeds, harvested by mowing.
- Morphine is the predominant alkaloid found in the varieties of opium poppy plant cultivated in most producing countries

- Poppies as medicine: Australia, Turkey and India are the major producers of poppy for medicinal purposes and poppy-based drugs, such as morphine or codeine

- In medicine, the term opiate describes any of the narcotic opioid alkaloids found as natural products in the opium poppy plant, as well as many semisynthetic chemical derivatives of such alkaloids

- There are a number of broad classes of opioids:
- Natural opiates: alkaloids contained in the resin of the opium poppy, primarily morphine, codeine, and thebaine, papaverine and noscapine
- Semi-synthetic opioids: created from the natural opiates, such as hydromorphone, hydrocodone, oxycodone, oxymorphone, desomorphine, diacetylmorphine (heroin), nicomorphine, dipropanoylmorphine, benzylmorphine and ethylmorphine;

- Fully synthetic opioids: such as fentanyl, pethidine, methadone, tramadol and dextropropoxyphene;
- Endogenous opioid peptides, produced naturally in the body, such as endorphins, enkephalins, dynorphins, and endomorphins.

PHARMACOLOGY

- Opioids bind to specific opioid receptors in the central nervous system and other tissues. There are three principal classes of opioid receptors, μ, κ, δ (mu, kappa, and delta), although up to seventeen have been reported, and include the ε, ι, λ, and ζ (Epsilon, Iota, Lambda and Zeta) receptors.

Dependence

- is characterised by extremely unpleasant withdrawal symptoms that occur if opioid use is abruptly discontinued after tolerance has developed. The withdrawal symptoms include severe dysphoria, sweating, nausea, rhinorrea, depression, severe fatigue, vomiting and pain.
- The acute withdrawal phase is often followed by a protracted phase of depression and insomnia that can last for months.

- **DIFFERENTIAL DIAGNOSIS**

1. OPIUM POISONING
2. ACUTE ALCOHOLIC POISONING
3. BARBITURATE POISONING
4. CARBOLIC ACID POISONING
5. CO POISONING
6. EPILEPTIC COMA
7. UREMIC COMA
8. DIABRTIC COMA
9. HYSTERICAL COMA
10. CEREBRAL HEMORRHAGE
11. BRAIN TRAUMA
12. CEREBRAL MALARIA
13. ENCEPHALITIS
14. MENINGITIS
15. HEAT HYPERPYREXIA

- Diacetylmorphine, or heroin, was first synthesized from morphine in 1874.
- It is formed simply by adding two acetyl groups. Heroin is around three times more potent than morphine. Its increased lipid solubility allows heroin to cross the blood-brain barrier more quickly. The drug is reconverted back to morphine before it binds to brain-tissue receptors.
- Heroin initially was marketed in 1898 by the Bayer Company of Germany. It was used as a cough remedy.
- Because morphine proved to be addictive, doctors began using heroin as a pain killer for surgery. However, heroin proved to be even more addictive than morphine–so much so that its use in medicine was stopped.
- And by 1925 heroin was branded as a dangerous drug nationally.

HEROIN

- WHITE BROWN BLACK TAR
- All these adulterated with quinine, lactose, mannitol
- Street heroin – Smack, junk, dope
- Heroin+ cocaine = SPEEDBALLS (alternatively known as **snowballing** or **powerballing**) most dangerous
- The combination is also known as **moonrocks** when smoked
- "Skin-popping" is injecting the drug just under the skin. This way it gets into the blood through tiny blood vessels.

- Heroin is the most fast-acting of all the opiates. When injected "**mainlining**", it reaches the brain in 7 seconds;
- Heroin (heated on a silver foil) and the smoke inhaled (**CHASING THE DRAGON**) reaches the brain in around 7-10 seconds. The peak is experienced in 10-15 min via this route
- The effects usually wear off in 3-5 hours, depending on the dose.
- Heroin is **not inherently toxic** to the organ systems of the body. Whereas a 200-400mg dose of heroin could kill a novice, a chronic user may take 1800mg without ill-effects

- Like alcohol, heroin is a depressant .
- But heroin differs from alcohol in 2 significant ways.
  1. It is not "organotoxic.“ That is why heroin dependency can last for years.

  2. An abuser usually does not die from the symptoms experienced from the withdrawal. The deaths associated with heroin are from overdosing rather than withdrawal.

- It is metabolised to acetylmorphine or monoacetylmorphine
- Monoacetylmorphine is then hydrolysed to morphine(half life 38 min)
- Chemical analysis will detect morphine and not heroin
- After injection Monoacetylmorphine and morphine are found in the urine almost immediately
- Tolerance occurs very rapidly (within days) and can be increased to more than 100 times the initial dose
- Notorious for causing sudden death
- Fatal dose 50 mg

- *SHORT-TERM EFFECTS*
- *"Rush"*
- *Depressed respiration*
- *Clouded Mental Functioning*
- *Nausea and Vomiting*
- *Suppression of Pain*
- *Spontaneous Abortion*

- With heroin, the rush is usually accompanied by a warm flushing of the skin, dry mouth, and a heavy feeling in the extremities, which may be accompanied by nausea, vomiting, and severe itching.
- After the initial effects, abusers usually will be drowsy for several hours.
- Mental function is clouded by heroin's effect on the central nervous system.
- Cardiac functions slow.
- Breathing is also severely slowed, sometimes to the point of death.

- ***LONG-TERM EFFECTS***
- Addiction
- Infectious Diseases (HIV/AIDS, Hepatitis B and C)
- Collapsed Veins
- Bacterial Infections
- Abscesses
- Infection of the Heart Lining and Valves
- Arthritis and Other Rheumatologic Problems
- One of the most detrimental long-term effects of heroin is addiction itself.

- Dependence

  heroin abusers gradually spend more and more time and energy obtaining and using the drug. Once they are addicted, the heroin abuser’s primary purpose in life becomes seeking and using drugs. The drug literally changes their brains.

Treatment

- Methadone 40 mg daily
- 80 mg in chronic addict
- Detoxification
- Narcotic antagonists such as naloxone, naltrexone, haloperidol, clonidine etc

BARBITURATES

- White, crystalline, odourless powder, with a bitter taste
- DEPRESSANTS

CLASSIFICATION

1. LONG ACTING: (Onset of action 2 hrs and duration of action 6-12 hrs)Barbitone, phenobarbitone, mephobarbitone, phenytoin
2. Intermediate acting: (Onset of action half -1 hrs and duration of action 3- 6 hrs) Amobarbitone, vinbarbital, allobarbitone
3. Short acting: (duration of action less than 3 hrs)Cyclobarbital, pentobarbital, amobarbital, seconal
4. Ultrashort acting: (Onset of action immediate and duration of action about 5-10 min)Pentothal sodium, kemithal sodium, thiamylal sodium

- Absorption, distribution and elimination
- **Barbiturates, Alcohol, CO produce irreversible brain damage and yet the patient survives for a sufficiently long period so that they are completely metabolised or excreted before death occurs**
- Action: CNS depression
- USES: Sedatives- hypnotics
  Anticonvulsants
  Induction of anaesthesia
  Antidote for convulsant poisoning

SIGNS AND SYMPTOMS

- Acute poisoning:
- CNS: Confusion, vertigo, ataxia, slurred speech, coma, Headache, paresthesia, Hypothermia is common
- Eye: pupils first constricted but later dilated
- CVS: Tachycardia, hypotension, circulatory failure
- Respiratory system: Slow, sighing respirations, progressing to respi failure
- Skin: Cold, clammy, cyanotic. Bullous lesions occur in a small % of comatose patients. Blisters (**barbiturate blisters**) on the skin often on an area of erythema strongly suggest barbiturate poisoning

- A stupor progressing to deep coma with inhibition of superficial and deep reflexes, and gradual loss of response to painful stimuli
- babinski toe sign may becomes positive
- Severe shock or respiratory failure are more common and more serious with medium and short acting barbiturates

CHRONIC POISONING

- due to regular intake of large doses (as in treatment of epilepsy, or in psychoneurotic patients) and is mainly characterized by somnolence, confusion, slurred speech, and the other S/S of Ch alcoholism etc.
- Tendon reflexes may be depressed, or there may be hypertonia and tremors of parkinsonian type
- Abrupt withdrawal can provoke an abstinence syndrome comprising anxiety, nausea, vomiting, agitation, confusion, tremor, ataxia, convulsions, delusion and hallucinations

FATAL DOSE

- short acting: 1-2 gms
- medium acting: 2-3 gms
- long acting : 3-5 gms
- The lethal blood levels are:
- SHORT ACTING: 3mg%
- MEDIUM ACTING: 7mg%
- LONG ACTING : 10 mg%
- FATAL PERIOD: 1-2 DAYS

TREATMENT

1. GL
2. MgSO4 left in the stomach to produce purgation
3. No specific antidote. Analeptics do not shorten the period of unconciousness or increase the rate of excretion
4. Scandinavian method: uses anti-shock measures, maintenance of patent airway, and adequate respi support
5. Hemodialysis and exchange transfusion are sometimes life saving
6. FAD: most useful in barbiturates which are not protein bound like phenobarbitone, allobarbitone, and barbitone

- Mannitol 100-200 ml of 25% solution, followed by an infusion of 500 ml of 5% sol during the next 3 hrs
- PM findings:

  Findings of asphyxia

  Fundus may be thickened, granular, eroded

  Hemorrhagic blistering and hemorrhagic necrosis of the gastric mucosa may be seen due to poisoning from seconal and Na amytal

  In delayed death there is symmetrical necrosis of the globus pallidus and corpus callosum, focal areas of necrosis in the cerebrum and cerebellum, and a variety of vascular lesions

MLI:

- Barbiturates commonly used for committing suicide
- Rarely used as homicidal poison
- Accidental poisoning may result due to “automatism” (involuntary suicide)

PSYCHEDELICS or Hallucinogens

- Alteration in environmental awareness while the individual maintains the capacity to recognise that what he is experiencing is not real
- Person is fully awake, alert, oriented but confronted with varied perceptual abnormalities and varied sensations.
- Synesthesias are frequent
- Euphoria or dysphoria, can be emotionally labile but realises that he is under the influence of drug
- LSD, mescaline, Dimethyltryptamine, psylocin, psylocybin, peyote, Phencyclidine(PCP)

PSYCHEDELICS or Hallucinogens

- Magic mushrooms
- MOA: Various neurotransmitters
- LSD involves the serotonin system and tropane alkaloids
- Psychc dependence but no abstinence

PSYCHEDELICS or Hallucinogens

- S/S
- Both sympathetic and parasympathetic symptoms are produced
- During hallucinations sensory perceptions are intensified
- COLOURS seem brighter and more clear