BLOOD TOXICITY

Lab 7

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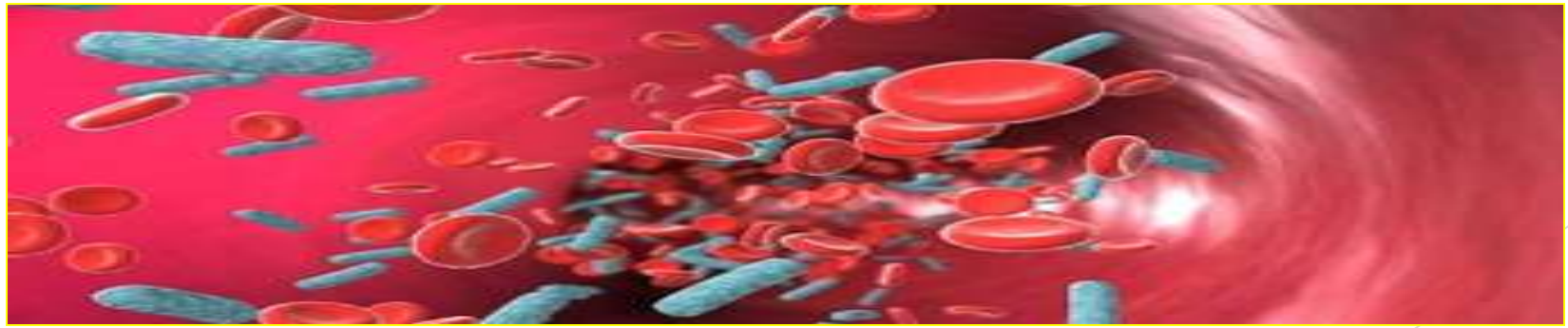

- **Hematotoxicology**: is the study of adverse effects of drugs, non-therapeutic chemicals and other agents in our environment on blood and blood-forming tissues.

Cyanide

- Cyanide is released from natural substances in some foods and in certain plants, including the seeds and some common fruits.
- In manufacturing, cyanide is used to make paper, textiles, and plastics. It is present in the chemicals used to develop photographs. Cyanide salts are used in metallurgy for electroplating, metal cleaning, and removing gold from its ore. Cyanide gas is used to exterminate pests and vermin in ships and buildings.

Deadly forms of cyanide include:

- sodium cyanide (NaCN)
- potassium cyanide (KCN)
- hydrogen cyanide (HCN)
- cyanogen chloride (CNCl)

Mechanism of toxicity:

- Cyanide inhibit mitochondrial cytochrome oxidase system (cytochrome AA3 complex) thus blocking ATP generation resulting in reduced cellular utilization of O2 & increased venous PO2 that reduce aerobic respiration with decreased pyruvate conversion in Krebs cycle leading to increased lactic acid formation & metabolic acidosis.

Toxicity:

- CN is one of the quickest acting poisons, inhibiting vital mitochondrial oxidation-reduction, resulting in loss of cellular energy generation thus affecting CNS & often producing respiratory paralysis & cardiac irregularities, but pulmonary failure usually precedes cardiac failure & causing death.
- Adult ingestion of as little as 200mg of NaCN or KCN may be fatal (Solutions of cyanide salts can be absorbed through intact skin), & 100mg/150lbs for HCN. Nevertheless, factors such as age, body mass, state of health, & mode of ingestion alter these values.
- CN produce histotoxic cellular hypoxia by initially binding to protein portion of cytochrome oxidase & then to ferric form of iron. Also binds to myoglobin in muscle & hemoglobin in blood especially the methemoglobin form.

Acute cyanide poisoning

- Acute cyanide poisoning is relatively rare, and the majority of cases are from unintentional exposure.
- When it does occur, symptoms are sudden and severe. You may experience:
- difficulty breathing
- seizure
- loss of consciousness
- cardiac arrest

Chronic cyanide poisoning

- Chronic cyanide poisoning can occur if you're exposed to 20 to 40 parts per million (ppm)Trusted Source of hydrogen cyanide gas over a substantial period of time.
- Symptoms are often gradual and increase in severity as time goes on.
- Early symptoms may include:
- Headache, drowsiness ,nausea ,vomiting, vertigo, bright red flush.

Additional symptoms may include:

- dilated pupils
- clammy skin
- slower, shallower breaths
- weaker, more rapid pulse
- convulsions

If the condition remains undiagnosed and untreated, it can lead to:

- slow, irregular heart rate
- reduced body temperature
- blue lips, face, and extremities
- coma
- death

Diagnosis

- Doctors may recommend Trusted Source several tests to determine the level of cyanide in the body, such as:
- **arterial blood gas**
- **urinalysis**
- **complete blood count**
- **chest X-ray**
- **EKG**
- Healthcare professionals may also recommend carboxyhemoglobin level tests if a person has been in a fire.
- Additionally, plasma lactate concentrations may determine whether a person has cyanide poisoning. Plasma lactate levels of more than 8 millimoles per liter Trusted Source are 70% specific for cyanide poisoning

Treatment:

- ## Supportive measures

- Maintain open airway & assist ventilation.
- Treat hypotension & seizure if occur.
- Start IV fluid & monitor vital signs & ECG closely.

- ## Decontamination

- Inhalation: remove victims from HCN exposure site & give supplemental O2.
- Skin: remove & isolate contaminated clothing & wash contaminated area with soap & water.
- Ingestion:
  - Prehospital: immediately administer activated charcoal
  - Hospital: immediately place gastric tube & administer activated charcoal.

- Hydroxocobalamin (Cyanokit): This is the preferred antidote, as it contains cobalt, which binds strongly to cyanide.
- Methemoglobin generators: These include amyl nitrite, sodium nitrite, and dimethyl aminophenol. They work by converting hemoglobin to a form that can compete with cyanide for binding.
- Sulfur donors: These include sodium thiosulfate and glutathione. They work by providing sulfur, which reacts with cyanide to form thiocyanate, a less toxic compound.