MERCURY POISONING

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Sources of Mercury Poisoning

Mercury is available as elemental mercury, inorganic mercury and organic mercury.

- **Sources**
  - Feed treated with mercurial fungicides, indiscriminate use of mercury containing drugs like ointments and diuretics, contaminated water, thermometers, mirror *etc*. serve as sources for mercury.
  - Mercury is also used in dental amalgams.
  - Mercury circulates in the environment because of its volatile nature and the earth continuously degases mercury.
  - Mercury is also released while burning coal.
  - Both acute and chronic mercury poisoning are rare because of the limited exposure to mercury.
  - More often, poisoning is due to consumption of obsolete mercurials.
  - Fish and other marine living organisms take up organic mercury from water and this mercury gets accumulated in these living things. This is known as bioaccumulation of mercury.
  - Though mercury is present only in small amounts in seawater, it is absorbed by the algae. It is efficiently absorbed, but only very slowly excreted by organisms.
  - Bioaccumulation and biomagnification result in build up in the adipose tissue of successive trophic levels: zooplankton, small nekton, larger fish *etc*.
  - Anything which eats these fish also consumes the greater level of mercury the fish have accumulated.
  - The consumers of such marine organisms are likely to have mercury poisoning.
  - Minamata disease sometimes referred to as Chisso-Minamata disease is a neurological syndrome caused by severe mercury poisoning.
  - Symptoms include ataxia, numbness in the hands and feet, general muscle weakness, narrowing of the field of vision and damage to hearing and speech.
  - In extreme cases, insanity, paralysis and death follow. This was first discovered in Minamata city in Japan in 1956. It was caused by the release of methyl mercury in the industrial waste water from a chemical factory. This highly toxic chemical bioaccumulated in shell fish and fish in Minamata Bay and the Shiranui sea.
  - When these were eaten, toxicity resulted in human, cat and dog. Toxic cases were reported for more than 30 years.

Absorption and Fate of Mercury

- Elemental mercury may become volatile and the mercury vapour, which is lipid soluble, can be absorbed by inhalation.
- Ingested elemental mercury and inorganic mercury salts are absorbed very slowly from the gastrointestinal tract.
- Organic mercurials are highly lipid soluble and are absorbed well from the gastrointestinal tract.
- Inorganic mercury salts are transported in erythrocytes and plasma.
- They accumulate in the renal cortex and localize in the lysosomes.
- Mercury easily crosses the blood brain barrier.
- Alkyl organic mercury compound accumulates in the brain.
- All forms of mercury can pass through the placental barrier and affect the foetus.
- Mercury in elemental form is oxidized to divalent mercury by catalases in tissues.
- Aryl mercurials are rapidly metabolized to inorganic salts. Alkyl mercurials are slowly metabolized to divalent mercury.
- Inorganic mercury is excreted mainly in urine, while organic mercury is excreted in faeces.

Mechanism of Toxicity of Mercury

- Inorganic mercury salts cause direct tissue necrosis and renal tubular necrosis.
- Mercuric ions bind covalently with sulphur and thereby inhibit sulphydryl enzymes in microsomes and mitochondria.
- Mercurial slats may also bind to proteins as mercaptides.
- Organic alkyl mercurials interfere with metabolic activity and prevent synthesis of essential proteins, leading to cellular degeneration and necrosis. Their most important target organ is the brain.

Clinical Symptoms and PM Lesions of Mercury Poisoning

Clinical symptoms

- Poisoning with inorganic mercury exhibits stomatitis, pharyngistis, vomiting, diarrhoea, dehydration and shock.
- Oliguria and azoturia are also observed. With organic mercurial toxicity, erythema of the skin, conjunctivitis, lachrymation, stomatitis and neurological seizures are reported.
- A condition known as mercurial ptyalism, has been reported in humans.The symptoms observed in this condition include profuse salivation, swelling of the gums, loosening of the teeth and necrosis of jaw bones.

Postmortem lesions

- Gastrointestinal ulcers, necrotic enteritis and colitis are noticed during post mortem.
- Pale and swollen kidney with renal tubular necrosis and fibrinoid degeneration of cerebral arterioles are also noticed.

Diagnosis and Treatment

Diagnosis

- Diagnosis is based on the level of mercury in the renal cortex, brain and liver.
- Differential diagnosis: Mercury poisoning should be differentiated from lead, thalliumand ethylene glycol poisoning, encephalitis, poliencephalomalacia and hog cholera erysipelas.

Treatment

- Treatment includes administration of egg white, activated charcoal, sodium thiosulphate (to bind mercury), saline cathartic and oral d-penicillamine.
- D-penicillamine is useful only if the gut is free of significant ingested mercury and only if the renal function is proper.
- BAL is not very effective after chronic exposure to organic mercurials.