Poisoning

GENERAL MANAGEMENT OF POISONING

⇒ MANAGEMENT OF POISONINING INVOLVES THE FOLLOWING STEPS:

» RESUSCITATION & INITIAL STABILISATION
» DIAGNOSIS OF TYPE OF POISON.
» PREVENTION OF FURTHER ABSORPTION OF POISON
» ADMINISTRATION OF ANTIDOTE.
» INCREASING THE CLEARANCE OF ABSORBED POISON.
» PREVENTION OF RECURRENCE OF POISONING.

I RESUSCITATION & INITIAL STABILISATION

(A, B & C) SHOULD BE ATTENDED

» AIRWAY
» BREATHING
» CIRCULATION

II DIAGNOSIS OF TYPE OF POISON

» THE DIAGNOSIS AND TREATMENT OF POISONS MUST PROCEED RAPIDLY WITHOUT WAITING FOR THE TOXICOLOGY SCREENING RESULTS.

III PREVENTION OF ABSORPTION OF POISON

EMESIS

» VOMITING CAN REMOVE UNABSORBED
» EMESIS CAN BE INDUCED BY DRINKING 200-400 ml OF A FULLY SATURATED SODIUM CHLORIDE SOLUTION.
» EMESIS IS LESS COMMONLY USED NOW, BECAUSE OF THE RISK OF ASPIRATION.

GASTRIC LAVAGE

» IT IS PERFORMED BY PASSING A WIDE-BORE NASOGASTRIC TUBE.
» PATIENT SHOULD BE IN LATERAL DECUBITUS POSITION WITH THE HEAD 15° TO 20° LOWER THAN FEET.

ACTIVATED CHARCOAL

» IT HAS AN EXTENSIVE NERWORK OF INTERCONNECTING PORES THAT BIND & TRAP CHEMICALS, THERE BY PREVENTING THEIR ABSORPTION & TOXICITY.
» IT IS USUALLY GIVEN AFTER GASTRIC LAVAGE.
» DOSE : 1g/kg BODY WEIGHT AS A SINGLE DOSE.

WHOLE BOWEL IRRIGATION (WBI)

» WBI REFERS TO THE ADMINATION OF LARGE VOLUMES OF A BALANCED ELETROCYTE SOLUTION WITH POLYETHYLENE GLYCOL, VIA A NASOGASTRIC TUBE, TO DECONTAMINATE THE GI TRACT.

IV ADMINISTRATION OF ANTIDOTE

» ANTIDOTES COUNTERACT THE EFFECTS OF POISONS BY NEUTRALIZING THEM OR BY ANTAGONIZING THEIR PHYSIOLOGIC EFFECTS.

» ANTIDOTES ARE AVAILABLE ONLY FOR A FEW POISONS.

POISON / DRUG	ANTIDOTE
ORGANOPHOSPHATES	ATROPINE , PAM , PRALIDOXIME
PARACETAMOL	N- ACETYLCYSTEINE
CYANIDE	DICOBALT EDETATE
METHANOL	ETHANOL
LEAD , ARSENIC , MERCURY	DIMERCAPROL (BAL)

V INCREASING THE CLEARANCE OF ABSORBED POISON

» ALKALINE DIURESIS

» HEMODYALYSIS

VI PREVENTION OF RECURRENCE OF POISONING

Composition:

>>COMPOSITION OF SNAKE VENOMS ARE COMPLEX MIXTURES OF ENZYMES, POLYPEPTIDES, GLYCOPROTEINS & METAL IONS.

HEMORRHAGINS
- CAUSE VASCULAR LEAKAGE & BLEEDING.

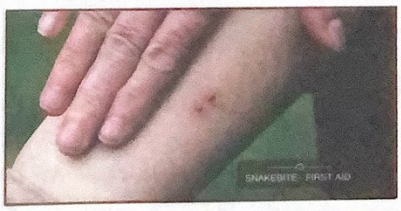
PROCOAGULANTS
- ACTIVATE CLOTTING FACTORS & CAUSE CONSUMPTIVE COAGULOPATHY.

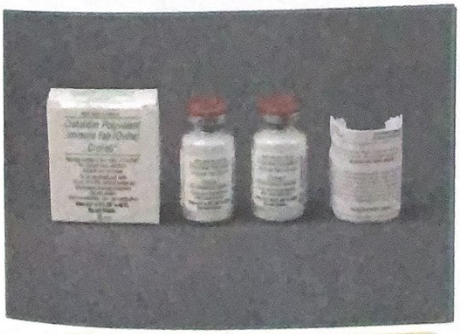
PROTEOLYTIC ENZYMES
- CAUSE LOCAL TISSUE NECROSIS,
  - COAGULATION DEFECTS
  - ORGAN DAMAGE.

CARDIOTOXINS
- MYOCARDITIS
- REDUCE CARDIAC OUTPUT.

NEUROTOXINS
- ACT AT PERIPHERAL NEUROMUSCULAR JUNCTIONS & INHIBIT NERVE IMPULSES.

MYOTOXINS
- LOCAL TISSUE NECROSIS
- RHABDOMYOLYSIS.





ANTIVENOM (10 VIALS USED)

Indications for Antivenom

>> **EVIDENCE OF SYSTEMIC ENVENOMATION**
- NEUROTOXICITY
- COAGULOPATHY
- RHABDOMYOLYSIS
- PERSISTENT HTN
- RENAL FAILURE

>> **EVIDENCE OF SEVERE LOCAL ENVENOMATION.**
- LOCAL TISSUE DESTRUCTION
- SWELLING
- PAIN

Management:

Field Management

R - REASSURE THE VICTIM
I - IMMOBILISE THE EXTREMITY
G - GET TO THE
H - HOSPITAL
T - INFORM THE PHYSICIAN OF TELLTALE SYMPTOMS & SIGNS.

Hospital Management

» BLOOD PRESSURE, HEART RATE, RESPIRATION, COAGULATION, RENAL, NEUROLOGICAL STATUS MUST BE MONITORED.

» ADMINISTRATION OF ANTI-SNAKE VENOM (ASV) - THIS IS THE MOST IMPORTANT STEP.
- SHOULD BE GIVEN AS SOON AS POSSIBLE.
- IT IS MOST EFFECTIVE IF GIVEN WITHIN 4 HRS OF BITE, BUT CAN BE GIVEN UPTO 24 HRS / LONGER
- ASV MAY BE MONOVALENT / POLYVALENT.

Dosage of ASV:

» LOCAL REACTION ONLY AT THE SITE OF BITE : 5 VIALS.
» LOCAL REACTION WITH SEVERE CELLULITIS : 5 - 15 VIALS.
» SEVERE REACTIONS WITH SYSTEMIC ENVENOMATION : 15 - 30 VIALS.

Supportive Measures

» IV FLUIDS
» INJ TETANUS TOXID - 0.5cc IM
» ANTIBIOTICS
» FRESH BLOOD / FFP TRANSFUSION
» NEOSTIGMINE 1mg IV STAT + ATROPINE 1mg IV
» ARTIFICIAL VENTILATION - FOR RESPIRATORY PARALYSIS.
» SURGICAL DEBRIDEMENT FOR LOCAL NECROSIS & SKIN GRAFTING.

ORGANOPHOSPHORUS POISONING

» THE MOST COMMON MODE OF POISONING IN INDIA IS WITH ORGANOPHOSHORUS (OP) COMPOUNDS BECAUSE OF THEIR WIDE AVAILABILITY.

» OP COMPOUNDS ARE WIDELY USED AS PESTICIDES IN AGRICULTURE.

» OP NERVE AGENTS ARE USED IN CHEMICAL WARFARE.

Mechanism of Toxicity

» OP COMPOUNDS ARE WELL ABSORBED THROUGH THE SKIN, LUNGS & GI TRACT.

→ INACTIVATE THE ENZYME ACETYLCHOLINESTERASE (AChE) BY PHOSPHORYLATION

↓ LEAD TO

ACCUMULATION OF ACh AT CHOLINERGIC SYNAPSES.

» LATER, THE ACETYLCHOLINESTERASE - OP COMPOUND UNDERGOES A CONFORMATIONAL CHANGE, KNOWN AS "AGING", WHICH RENDERS THE ENZYME IRREVERSIBLY RESISTANT TO REACTIVATION BY OXIMES.

Clinical features :

3 PHASES :

1. ACUTE CHOLINERGIC PHASE
2. INTERMEDIATE SYNDROME (IMS)
3. ORGANOPHOSPHATE - INDUCED DELAYED POLYNEUROPATHY (OPIDN).

Acute Cholinergic Phase

» DUE TO ACETYLCHOLINE EXCESS AT THE SYNAPSES.

» SYMPTOMS USUALLY ONE HOUR OF EXPOSURE.

MUSCARANIC

D – DEFECATION
U – URINATION
M – MIOSIS
B – BRONCHORRHEA
– BRONCHOSPASM
– BRADYCARDIA
E – EMESIS
L – LACRIMATION
S – SALIVATION

NICOTINIC

» FASCICULATIONS
» MUSCLE WEAKNESS
&
» PARALYSIS

CNS MANIFESTATIONS

» ANXIETY
» RESTLESSNESS
» UNCONSCIOUSNESS
» CONVULSIONS

Intermediate Syndrome (IMS)

»THIS BEGINS 1 TO 3 DAYS AFTER EXPOSURE.

»USUALLY OCCURS AFTER THE ACUTE CHOLINERGIC PHASE, MAY ALSO OCCUR ALONG WITH IT.

»POSTULATIONS ABOUT THE MECHANISM OF IMS:

- PERSISTENCE OF NICOTINIC EFFECTS DUE TO LACK OF EARLY USE OF OXIMES
- RELEASE OF ORGANOPHOSPHATES FROM THE ADIPOSE TISSUE ACTING ON THE NICOTINIC RECEPTORS.
- NEUROMUSCULAR JUNCTION DYSFUNCTION.

»MANIFESTATIONS:

- WEAKNESS OF NECK MUSCLES
- ↓SED DEEP TENDON REFLEXES
- CRANIAL NERVE ABNORMALITIES
- PROXIMAL & RESPIRATORY MUSCLE WEAKNESS / PARALYSIS.

»MUSCURANIC SIGNS ARE NOT SEEN USUALLY.

Organophosphate - induced delayed neuropathy

» THIS OCCURS 1 - 3 WEEKS AFTER ACUTE OP EXPOSURE.

» DUE TO DEGENERATION OF LONG MYELINATED NERVE FIBRES.

» PATIENTS PRESENTS WITH TRANSIENT, PAINFUL "STOCKING - GLOVE" PARESTHESIAS FOLLOWED BY A SYMMETRICAL MOTOR POLY NEUROPATHY (FLACCID WEAKNESS OF LOWER LIMBS).

» RECOVERY FROM OPIDN IS USUALLY INCOMPLETE.

» CRAMPS IN THE LEGS, NUMBNESS, PARASTHESIA IN THE DISTAL UPPER & LOWER LIMB.

Diagnosis:

» HISTORY & EXAMINATION FINDINGS, GARLIC LIKE ODOUR.

» PLASMA CHOLINESTERASE LEVELS ARE REDUCED TO LESS THAN 50% OF NORMAL

» TOXICOLOGY - GASTRIC CONTENTS IF POSSIBLE.

Management:

GENERAL MEASURES

» FURTHER EXPOSURE IS PREVENTED BY REMOVING THE COTAMINATED CLOTHES.

» PATIENT ADMITTED TO ICU & VITAL PARAMETERS ARE MONITORED.

» OXYGEN

» GASTRIC LAVAGE

» ACTIVATED CHARCOAL

» IV FLUIDS

» ENDOTRACHEAL INTUBATION & VENTILATOR SUPPORT.

SPECIFIC MEASURES

» ATROPINE - ANTAGONISES THE MUSCURANIC EFFECTS OF ACh.
- IT DOES NOT REVERSE NICOTINIC EFFECTS SUCH AS MUSCLE FASCICULATION.

» OXIMES - PRALIDOXIME (PAM)
- OBIDOXIME
- THESE ARE CHOLINESTERASE REACTING AGENTS & ARE EFFECTIVE IN TREATING BOTH MUSCURANIC & NICOTINIC EFFECTS OF OP COMPOUND.

» MAGNESIUM SULPHATE

» INTERMEDIATE SYNDROME IS TREATED BY VENTILATOR SUPPORT.

» THERE IS NO SPECIFIC THERAPY FOR OPIDN. REGULAR PHYSIOTHERAPY MAY REDUCE DEFORMITIES & MUSCLE WASTING.

BARBITURATE POISONING

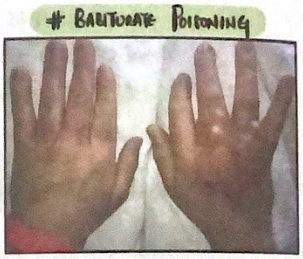
Definition :

>> SEDATIVE - HYPNOTICS ARE A GROUP OF DRUGS THAT CAUSE CNS DEPRESSION.
>> THESE DRUGS INCLUDE - BENZODIAZEPINES
- BARBITURATES
- SLEEPING PILLS SUCH AS ZOLPIDEM & ZALEPLON.

Mechanism

>> ALL THE SEDATIVE - HYPNOTICS ARE GENERAL CNS DEPRESSANTS.
>> MOST STIMULATE THE ACTIVITY OF GABA, AN INHIBITORY NEUROTRASMITTER IN CNS.

Clinical features : SIRPH NIBBI •̀_•́


Barbiturate Poisoning

S - SLURRED SPEECH
I - INCORDINATION & UNSTEADY GAIT
R - RESPIRATORY DEPRESSION
P - PSYCHHATRIC MANIFESTATIONS (INAPPROPRIATE BEHAVIOR, LABILE MOOD, IMPAIRED JUDGEMENT & SOCIAL FUNCTIONING)

H - HYPOTENSION
N - NYSTAGMUS
I - IMPAIRED ATTENTION / MEMORY
B - BRADYCARDIA
B - BULLOUS SKIN
I - IMPAIRED CONSCIOUSNESS.

Investigations :

>> COMPLETE BLOOD COUNT (CBC).
>> ARTERIAL BLOOD GAS (ABG)
>> BLOOD GLUCOSE, ELECTROLYTES
>> ECG
>> TOXICOLOGY SCREEN.

Management :

GENERAL MEASURES

>> FURTHER EXPOSURE IS PREVENTED BY REMOVING THE COTAMINATED CLOTHES.
>> PATIENT ADMITTED TO ICU & ULTAL PARAMETERS ARE MONITORED.
>> OXYGEN
>> GASTRIC LAVAGE IS NOT ADVISED IN PURE BENZODIAZEPINE OVERDOSE.
>> ACTIVATED CHARCOAL, REPEATED DOSES ARE NECESSARY
>> IV FLUIDS
>> ENDOTRACHEAL INTUBATION & VENTILATOR SUPPORT.

SPECIFIC MEASURES

>> BENZODIAZEPINES - FLUMAZENIL IS A BENZODIAZEPINE ANTAGONIST.
>> BARBITURATES - ALKALINE DIURESIS & HEMODIALYSIS.

Mechanism of Toxicity

>> AFTER INGESTION, **ALUMINIUM PHOSPHIDE** REACTS WITH WATER IN THE STOMACH TO RELEASE PHOSPHINE GAS WHICH HAS LOCAL AS WELL AS SYSTEMIC TOXICITY.

>> **LOCAL EFFECTS** - SEVERE BURNING RETROSTERNAL PAIN & VOMITING

>> **SYSTEMIC TOXICITY** - OCCURS AFTER ABSORPTION FROM GI TRACT.

ALUMINIUM PHOSPHIDE
↓ ⊖ INHIBITS
CYTOCHROME C OXIDASE
↓
EFFECTS ON MITOCHONDRIA
↓
CELLULAR HYPOXIA
↓
ORGAN DAMAGE

Diagnosis :

>> DETECTING PHOSPHINE IN THE EXHALED AIR / STOMACH ASPIRATE BY USING SILVER-NITRATE - IMPREGNATED STRIP / GAS CHROMATOGRAPHY.

Management :

>> **GASTRIC LAVAGE** - WITH $KMnO_4$ / $MgSO_4$ / COCONUT OIL.

>> **ACTIVATED CHARCOAL**

>> **ANTACIDS / H_2 BLOCKERS** - TO REDUCE BURNING PAIN IN THE STOMACH.

>> **MAGNESIUM SULFATE ($MgSO_4$)** - VERY EFFECTIVE IN COUTERACTING THE TOXIC EFFECTS OF ALUMINIUM PHOSPHATE.

>> **SODIUM BICARBONATE INFUSION** - TO CORRECT METABOLIC ACIDOSIS.

>> **MORTALITY IS HIGH** & MOST PATIENTS DIE DESPITE OPTIMAL SUPPORTIVE CARE.

PARACETAMOL POISONING

>> IT IS COMMON BECAUSE OF ITS **WIDE AVAILABILITY AS AN OVER-THE-COUNTER DRUG.**
>> MAXIMUM RECOMMENDED DAILY DOSE IS **4g IN ADULTS.**
>> TOXICITY IS LIKELY TO OCCUR AT DOSES **>250 mg/kg PER DAY.**
>> ALMOST ALL PATIENTS WHO INGEST >350 mg/kg DEVELOP SEVERE LIVER TOXICITY.

Mechanism of Toxicity

>> PARACETAMOL IS **METABOLISED IN THE LIVER** TO NON-TOXIC COMPOUNDS.
>> IN ACUTE OVERDOSE, METABOLISM BY CONJUGATION BECOMES SATURATED & EXCESS PARACETAMOL IS OXIDATIVELY METABOLISED BY THE CYP ENZYMES TO THE HEPATOTOXIC METABOLITE, N-ACETYL-P-BENZOQUINONE IMINE (NAPQI)

Clinical features :

>> **INITIAL COMPLAINTS** ARE
- NAUSEA
- VOMITING
- DIAPHORESIS
- PALLOR
- LETHARGY
- MALAISE

>> **LIVER DAMAGE** USUALLY DEVELOPS 1-3 DAYS AFTER INGESTION.

- JAUNDICE
- HEPATIC ENCEPHALOPATHY
- HYPERAMMONEMIA
- BLEEDING DIATHESIS
- MARKED ELEVATION OF LIVER ENZYMES.

>> RARELY, **RENAL FAILURE & ACUTE PANCREATITIS MAY OCCUR.**

Investigations :

>> CONFIRMATION OF DIAGNOSIS & SEVERITY OF POISONING CAN BE ASSESSED BY MEASUREMENT OF SERUM PARACETAMOL LEVELS.

Management :

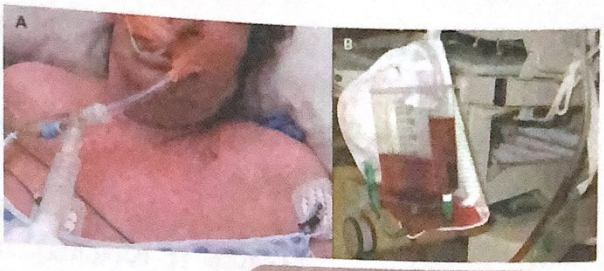
>> GASTRIC LAVAGE
>> ACTIVATED CHARCOAL
>> N-ACETYLCYSTEINE (NAC) IS THE ANTIDOTE.
>> METHIONINE, IF NAC IS NOT AVAILABLE.
>> LIVER TRANSPLANTATION.

CYANIDE POISONING

» CYANIDE IS AMONG THE MOST RAPIDLY LETHAL POISONS KNOWN TO MAN.
» IT CAUSES DEATH WITHIN MINUTES TO HOURS OF EXPOSURE.

Etiology :

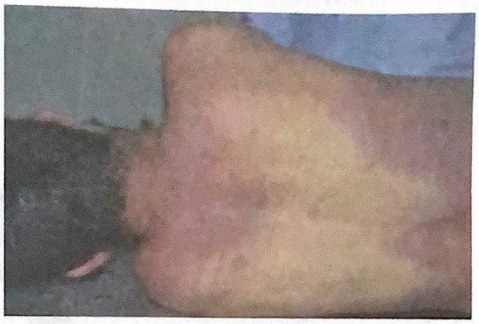
» SMOKE INHALATION
» SUICIDAL INGESTION
» INDUSTRIAL EXPOSURES
» INHALATION OF HYDROCYANIC ACID
» INGESTION OF INORGANIC CYANIDE SALTS
» CYANAMIDE (RELEASES CYANIDE).
» AMYGDALA FROM BITTER ALMONDS.



CYANIDE - CHERRY RED

Mechanism of Toxicity

CYANIDE
↓
CYTOCHROME OXIDASE
↓
STOPPAGE OF OXIDATIVE PHOSPHORYLATION.
↓
HISTOTOXIC ANOXIA
↓
CELLULAR DYSFUNCTION & DEATH



POSTMORTEM CHANGE

Clinical features :

» THERE MAY BE CHARACTERISTIC SMELL OF BITTER ALMONDS.
» AFTER INHALING CYANIDE, THERE IS HEADACHE, ANXIETY, NAUSEA & A METALLIC TASTE.
» EYE & MUCOUS MEMBRANE IRRITATION, BRONCHORRHEA, COUGH & DYSPNEA.
» INGESTION OF CYANIDE SALTS RESULTS IN GASTRIC IRRITATION CAUSES VOMITING & ABD PAIN.
» MULTIORGAN FAILURE - RENAL FAILURE, HEPATIC NECROSIS, PULM. EDEMA, RHABDOMYOLYSIS.
» SKIN APPEARS FLUSHED & CHERRY-RED DUE TO NON-UTILISATION OF OXYGEN BY CELLS.
» CONVULSIONS, COMA & DEATH OCCURS WITHIN A FEW HOURS.

Investigations :

» ARTERIAL & VENOUS BLOOD GASES
» BLOOD LACTATE LEVEL
» RBC / PLASMA CYANIDE CONCENTRATION
» METHEMOGLOBIN LEVEL
- PRESENCE OF METHEMOGLOBIN IN THE BLOOD IS REASSURING BECAUSE IT INDICATES THAT THERE IS NO FREE CYANIDE AVAILABLE FOR BINDING, BECAUSE METHEMOGLOBIN VIGORDUSLY BINDS CYANIDE TO FORM CYANOMETHEMOGLOBIN.

Treatment :

» GASTRIC LAVAGE
» ACTIVATED CHARCOAL
» HYDROXOCOBALABIN - A PRECURSOR OF VIT-B_{12}, CONTAINS A COBALT MOEITY WHICH BINDS TO CYANIDE, FORMING CYANOCOBALAMINE.
» TAYLOR CYANIDE ANTIDOTE PACKAGE - IT CONTAINS
- AMYL NITRITE
- SODIUM NITRITE
- SODIUM THIOSULFATE

» DICOBALT EDETATE

ARSENIC POISONING

» ARSENIC IS A METALLOID ELEMENT. IT IS TASTELESS, ODORLESS, & WELL ABSORBED AFTER INGESTION / INHALATION.

» COMMON SOURCES OF EXPOSURE ARE GROUND WATER & FOOD WITH ARSENIC CONTENT.

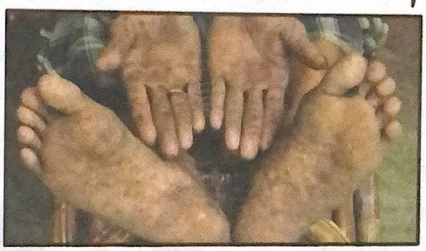
» IN EXPOSED INDIVIDUALS, HIGH CONCENTRATIONS OF ARSENIC ARE PRESENT IN BONE, HAIR & NAILS.

Mechanism of Toxicity

» ARSENIC INHIBITS THE ENZYME PYRUVATE DEHYDROGENASE COMPLEX, WHICH CATALYSES THE OXIDATION OF PYRUVATE TO ACETYL-COA.

» THIS LEADS TO DISRUPTED THE ENERGY SYSTEM OF THE CELL RESULTING IN CELL DEATH.

Clinical features :



Acute Poisoning

» THIS CAN OCCUR AFTER INGESTION / INHALATION OF ARSENIC DUSTS / FUMES.

» SYMPTOMS MAY DEVELOP WITHIN MINUTES / HOURS.

» INITIALLY GI SYMPTOMS ARE SEEN
- NAUSEA
- VOMITING
- ABD PAIN
- DIARRHEA
- GARLIC ODOR OF THE BREATH & STOOL.

» THESE ARE FOLLOWED BY -DEHYDRATION
-HYPOTENSION
-IRREGULAR PULSE
-CARDIAC INSTABILITY

» ACUTE ENCEPHALOPATHY MAY DEVELOP & LEAD TO DELIRIUM, COMA & SEIZURES.

» RENAL INJURY CAN LEAD TO - PROTEINURIA
- HEMATURIA
-ACUTE TUBULAR NECROSIS.

» IN SEVERE CASES, SHOCK, ARDS & DEATH MAY OCCUR.

Chronic Poisoning

» CHRONIC ARSENIC EXPOSURE FROM DRINKING WATER HAS BEEN REPORTED IN MANY PARTS OF THE WORLD

» MANIFESTATIONS ARE PERIPHERAL NEUROPATHY, SKIN ERUPTIONS, HEPATOTOXICITY, BONE MARROW DEPRESSION & ↑SED RISK OF CANCERS. » MEE'S LINES MAY BE SEEN IN SOME.

Management :

» ELIMINATION OF FURTHER EXPOSURE.

» GASTRIC LAVAGE & ACTIVATED CHARCOAL IN CASES OF INGESTION.

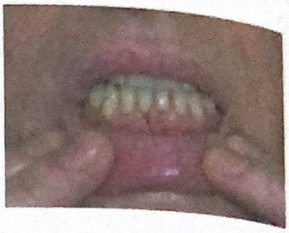
» CHELATION IS INDICATED IN PATIENTS WITH SYMPTOMATIC ARSENIC POISONING.

» DIMERCAPROL & SUCCIMER.

LEAD POISONING

» LEAD EXPOSURE CAN OCCUR IN NUMEROUS WORK SETTINGS, SUCH AS

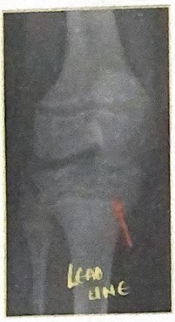
MANUFACTURING / USE OF
- BATTERIES,
- SOLDER,
- AMMUNITIONS
- PAINT
- CAR RADIATORS
- CABLE & WIRES
- SOME COSMETICS
- CERAMIC WARE
- PLUMBING



» LEAD IS ALSO FOUND IN SOME TRADITIONAL HISPANIC & AYURVEDIC MEDICINES.

» LEAD CONTAINING BULLETS LODGED IN THE BODY CAN RESULT IN CHRONIC LEAD TOXICITY.

Mechanism of Toxicity



» LEAD IS ABSORBED FROM THE LUNGS / GI TRACT.
» IN ADULTS, ABSORPTION OF LEAD VIA THE RESP. TRACT IS MORE SIGNIFICANT ROUTE OF ENTRY.
» GI ABSORPTION IS THE PREDOMINANT ROUTE IN CHILDREN.
» ONCE ABSORBED, LEAD IS DISTRIBUTED TO THE BLOOD, SOFT TISSUES & SKELETON.

LEAD
- INHIBITS SULFHYDRYL - DEPENDENT ENZYMES SUCH AS GAMMA - AMINOLEVULINIC ACID DEHYDRATASE (ALA-D) & FERROCHELATASE.
- INTERFERES WITH MITOCHONDRIAL RESPIRATION & VARIOUS NERVE FUNCTIONS.
- CAN ALSO AFFECT DNA & RNA.

Clinical features :

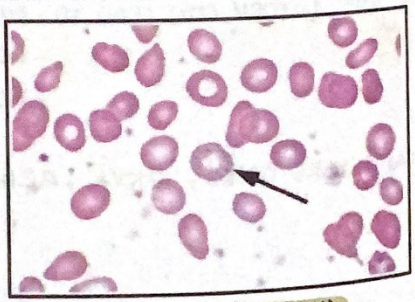
» COLICKY ABD PAIN
» CONSTIPATION
» JOINT PAINS
» MUSCLE ACHES
» HEAD ACHE
» ANOREXIA
» DECREASED LIBIDO
» DIFFICULTY CONCENTRATING
» DEFICITS IN SHORT-TERM MEMORY
» ANEMIA
» NEPHROPATHY
» COMA & CONVULSIONS MAY OCCUR IN SEVERE POISONING.
» A BLUISH LEAD LINE MAY BE SEEN AT THE GUM-TOOTH LINE.
» CHRONIC LEAD POISONING CAN CAUSE LEARNING DISORDERS & MOTOR NEUROPATHY

Diagnosis :

» WHOLE BLOOD LEAD LEVELS ABOVE 10/dl
» MICROCYTIC ANEMIA WITH BASOPHILIC STIPPLING
» FREE ERYTHROCYTE PROTOPORPHYRIN ↑↑↑.
» X-RAY FLUORESCENCE

Treatment :

» MAINTAIN AIRWAY & TREAT COMA & CONVULSIONS IN SEVERE POISONING.
» AVOID FUTURE EXPOSURE
» CHELATION THERAPY



BASOPHIL STIPPLI

HEAT STROKE

Definition :

» HEAT STROKE IS DEFINED AS **A CORE BODY TEMPERATURE IN EXCESS OF 40°C WITH ASSOCIATED CNS DYSFUNCTION** IN THE SETTING OF A LARGE ENVIRONMENTAL HEAT LOAD THAT CANNOT BE DESSIPATED.

» IT RESULTS FROM
- A COMPLETE BREAKDOWN OF THE THERMOREGULATORY MECHANISM
- COMPLETE FAILURE OF SWEATING.
- CELLULAR DAMAGE OF VITAL ORGANS.

Clinical features :

» THERE ARE TWO TYPES OF HEAT STROKE; **EXERTIONAL** & **NON-EXERTIONAL**.

OCCURS IN HEALTHY INDIVIDUALS THAT ENGAGE IN HEAVY EXERCISE DURING HIGH TEMPERATURE

OCCURS IN INDIVIDUALS WITH AN UNDERLYING DISORDER SUCH AS MENTAL ILLNESS, HYPERTHYROISM, OBESITY, EXTREMES OF AGE & USE OF DRUGS.

» **INITIAL MANIFESTATIONS** - FAINTNESS
- DIZZINESS
- VERTIGO
- NAUSEA
- ABD. PAIN

» **CNS MANIFESTATIONS** - ALTERED SENSORIUM
- SEIZURES
- COMA

» **TACHYPNEA, BIBASAL LUNG CREPITATIONS** DUE TO ARDS.

» **SKIN** IS - FLUSHED
- HOT
- DRY

» **JAUNDICE & PETECHIAE**

» **EXCESSIVE BLEEDING** DUE TO DIC.

» **RECTAL TEMP ≥40°C**

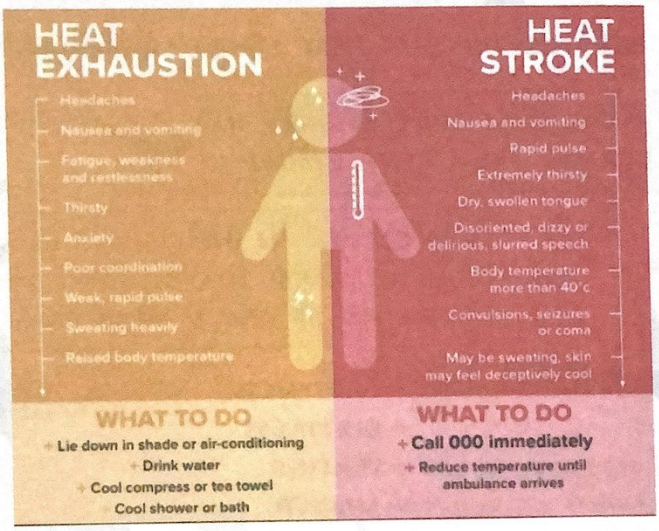
» **DEATH** - DUE TO ARDS, DIC, MI, ACUTE ADRENAL INSUFFICIENCY.

Laboratory Features :

- HEMOCONCENTRATION
- LEUKOCYTOSIS
- ELEVATED UREA & CREATININE
- PROTEINURIA
- COAGULOPATHY & DIC
- ELEVATED LIVER ENZYMES
- RESPIRATORY ALKALOSIS & METABOLIC ACIDOSIS.

} SHOWN BY INVESTIGATIONS

- ECG MAY SHOW NON-SPECIFIC ST DEPRESSION & T-WAVE INVERSION.



Treatment :

- 100% OXYGEN
- ENDOTRACHEAL INTUBATION & VENTILATORY SUPPORT
- REDUCTION OF BODY TEMPERATURE
- HYDROCORTISONE / DEXAMETHASONE INJECTION IV.
- RENAL FAILURE MAY REQUIRE HEMODIALYSIS.