4) Poisoning in Children – Dr. Jamal

- 60% of all poisoning occur in children younger than 6 years.
- 95% of all poisoning children are accidental.
- Most poisons are ingested; some may be inhaled, spilled on to skin or in to the eyes or rarely injected.

The most common agents involved in accidental poisoning include:

- Kerosene
- Cleaning agents
- CO
- Prescription medications

Most children with poisoning present to hospital without a specific history of toxic exposure, that’s why poisoning as a cause for various signs & symptoms must always be kept in mind & asked for in detail.

Features of poisoning in general include: convulsion, vomiting & diarrhea, altered mental state & behavior, acidosis, dysrhythrias, abnormal odor, abnormal skin color, pupil changes……etc.

The basic historical information including:

- Confirmation of poisoning
- Identification of the specific agent
- Route & quantity exposed to
- Time of the exposure
- Any symptoms occurred before arrival
- Any preexisting medical conditions & the use of therapeutic medications.

Even if the exact nature of the exposed poison is not determined in the history, many poisons show characteristic odor & or signs which help in making a possible diagnosis on which base antidotes can be given & toxicological tests carried out.

**Among the symptoms with which some poisoned patient may present are:**

1/ coma: mainly caused by

- Antihistaminic, sedatives & hypnotics, CO, cyanide, organophosphates, tricyclic antidepressants.
- The most common cause of death in comatose poisoned patient is acute respiratory arrest, thus the importance of clear airway, assisted ventilation & O2. When comatose poisoned patients are evaluated, other causes of coma such as head trauma, hypoglycemia, hypothermia, meningitis & encephalitis should be looked for.

2/ convulsion: causes of which include

- Anticholinergics, CO, phenothiazines, tricyclic antidepressants, B-blockers.
- As poisoning is a relatively uncommon cause of convulsion in children, other causes must always be considered including hypoxia, hypoglycemia, hyponatremia, head injury, C.N.C infection & febrile seizure. Convulsions cause by toxins is usually more difficult to control.
3/ cardiac arrhythmias: caused by
- B-blockers, calcium channel blockers, digoxin, tricyclic antidepressants, amphetamines, phenothiazines, cyanide.
- Proper ECG record is part of the initial evaluation of all poisoned patient. Sinus tachycardia is a common & nonspecific finding in various poisoning.
- Prolonged Q-T interval occurs in phenothiazine poisoning, widened QRS is a feature of tricyclic antidepressants.
- Treatment of arrhythmias depends on the specific rhythm present.

4/ GI symptoms: caused by;
- Arsenic, iron, mercury, poisonous mushrooms & lithium
- Feature of GI symptoms include nausea, vomiting, abdominal colic, diarrhea & GI hemorrhage. These may be due to direct effect of the toxin on GI or is a systemic effect after absorption of the toxin. A single episode of vomiting is quite possible in any ingested poisoning.

5/ metabolic acidosis: toxin causes of which include
- CO, cyanide, ethanol, ethylene glycol, iron, methanol & salicylates.
- Persistent unexplained metabolic acidosis may be the only initial clue to a poisoning. Evaluation of metabolic acidosis should include estimation of blood gases, serum electrolytes, blood urea, blood glucose & serum osmolality. Calculation of the anion gap (A.G.) is helpful in the differential diagnosis of metabolic acidosis.
  - A.G. = S.Na (mEq/L) \_ [S.Cl (mEq/L) + S.HCO3 (mEq/L)]
  - Normal A.G. is 8-12 mEq/L
  - Serum osmolality should both be measured directly & calculated as follows:
  - Calculated Serum osmolality=
  - 2 [Na (mEq/L)] +B.U (mg/dl)/2.8+B.glucose (mg/dl)/18
  - A measured serum osmolality which is more than the calculated one by more than 10mOsm indicates presence of some somatically active substances that are not accounted by the calculation formula. These substances include ethanol, methanol, alcohol & ethylene glycol.

On physical examination selective findings in poisoned patient include:
1/ special odors: e.g. bitter almond in cyanide, garlic in arsenic & organophosphorous, & acetone in salicylate.

2/skin findings such as:
- Cherry red color in CO & cyanide
- Sweaty in organophosphorous & sympathomimetics
- Dry skin in anticholinergics
- Urticaria as an allergic reaction
- Gray cyanosis in methemoglobinemia

3/Eye findings:
- **Meiosis:** opiates, organophosphorous & phenothiazines
- **Mydriasis:** atropine, amphetamine & tricyclic antidepressants
- **Nystagmus** in dilantin
- **Retinal hemorrhage** in CO & methanol

4/ Fever: tricyclic antidepressants, salicylates, thyroxin, anticholinergics, amphetamine & theophylline.
Laboratory evaluations of a potentially significant poisoning include:

- C.B.C., B.U. & electrolytes, blood sugar, blood gases & osmolality.
- ECG, chest x-ray for aspiration _+ pulmonary edema.
- Abdominal X-ray may visualize radiopaque substances as iron, heavy metals, chloral hydrate & some phenothiazines.
- Routine toxicology screen, unless the toxin has been identified from history, physical exam. & some routine laboratory tests, is expensive, time consuming & subject to false +ve & false –ve results. However specific quantitative level estimation of certain drugs may be useful in determining the need for a specific intervention or antidote.

General management principles of poisoning

1. Ensure adequate breathing & circulation by positioning, suctioning, O2, intubation & ventilation.
2. Establish I.V access & give boluses of normal saline + dextrose 20ml/kg as needed to improve peripheral perfusion & stabilize the vital signs. Inotrope (dopamine or dobutamine) may be considered. Blood pressure is checked frequently. Correct hypoglycemia with 5ml/kg of 10% dextrose.
3. Monitor conscious state by GCS, pupillary light reflexes. In presence of depressed consciousness give 2mg of naloxone (narcan) I.V regardless of the age. Toxicin induced convulsions can be difficult to control. In general diazepam, lorazepam (may induce further respiratory depression requiring ventilation) or phenytoin. Antibiotics are not given unless secondary bacterial infections are present.
4. Removal of the poison:
   a. Body washes & eye cleaning in special poisoning e.g. organophosphates.
   b. Activated charcoal (ACC): is effective in decreasing absorption of most ingested poisons except in iron, lithium, alcohol, arsenic, acids, alkali, lead, hydrocarbons & cyanide. It has a very strong adsorbent power; it is given in a dose of 1gm/kg mixed with water orally or through an N.G.T during the 1st hour after ingestion. As charcoal interferes with visualization during endoscopy, it is not used in caustic ingestion. Activated charcoal also adsorbs some drugs directly from the blood perfusing GIT in a process referred to as GI dialysis. It is usually given after emesis or gastric lavage. The dose can be repeated every 2-4 hours until the first charcoal stool appears.
   c. Ipecac syrup is a very strong emetic acts by both direct gastric irritation & central chemoreceptor stimulation. It can only be effective if given within the 1st 30 min. of ingestion. It is contraindicated in comatose patient, caustic or hydrocarbon ingestion & in children younger than 6 months. It is rarely used nowadays.
   d. Gastric lavage: this is performed using a large bore orogastric tube & irrigating the stomach by normal saline, during the 1st 2hours of ingestion (up to 12 hours in salicylate, opiates & Tricyclic antidepressants poisoning in which there is delayed gastric emptying). Lavage is contraindicated in:
      i. Caustic ingestion
      ii. Nontoxic ingestion
      iii. Hydrocarbons
      iv. Delayed presentation
      v. Comatose patient(except when the ingested substance is highly toxic & the airways can be secured)
   e. Whole bowel irrigation (WBI): means nearly complete emptying of the bowel using polyethylene glycol (an osmotic agent).This method is effective in iron & other heavy metal poisoning & in sustained released medications.
   f. Use of antidotes: a relatively small number of toxic substances have antidotes e.g.
<table>
<thead>
<tr>
<th>Toxin</th>
<th>Antidote</th>
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<tbody>
<tr>
<td>Acetaminophen</td>
<td>N-acetylcysteine</td>
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<td>Anticholinergics</td>
<td>Physostigmine</td>
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<td>Benzodiazepines</td>
<td>Flumazenil</td>
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<td>CO</td>
<td>O₂</td>
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<td>Scorpion &amp; snake poisons</td>
<td>Specific antivenom</td>
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<tr>
<td>Cyanide</td>
<td>Amylnitrite, Na nitrite, Na thiosulphate &amp; B&lt;sub&gt;1₂&lt;/sub&gt;</td>
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<td>Heavy metals</td>
<td>D-Penicillamine</td>
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<tr>
<td>Dystonia inducing toxins</td>
<td>Diphenhydramine</td>
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<td>Methemoglobinemia inducing toxins</td>
<td>Methylene blue</td>
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<td>Iron</td>
<td>Deferoxamine</td>
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<td>INH</td>
<td>B&lt;sub&gt;6&lt;/sub&gt;</td>
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<td>Methanol &amp; ethylene glycol</td>
<td>Ethanol</td>
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<td>Narcotics</td>
<td>Naloxone</td>
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<td>Organophosphate comp. or Carbamate insecticides)</td>
<td>Atropine</td>
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<td>B-blockers</td>
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<td>Calcium channel blockers</td>
<td>Calcium &amp; glucagon</td>
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<td>Tricyclic antidepressants</td>
<td>Bicarbonate</td>
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**g. Enhancing excretion:**

i. Forced diuresis for toxins which are mainly excreted by kidney. 2-3 times the maintenance I.V. fluids are given to establish a urine O.P. of 2-5 ml/kg/hr. with addition of 50-75 Meq/L NaHCO₃ + concomitant use of furosemide or mannitol.

ii. Hemodyalisis especially in salicylate, ethanol & theophylline poisoning.

iii. Exchange transfusion especially in neonates & infants with hemolysis or methemoglobinemia.

iv. Hemoferfusion.

v. Cathartics can be used to hasten emptying of GIT.

**Prevention of poisoning:**

1. Protection of the child from poisoning agent by:
   a. Keep the poisoning substances out of reach of children.
   b. There should be a proper secured place at home for potentially poisonous substances.
   c. Don’t put poisonous substances in containers otherwise used for ordinary foods as water.
   d. All drugs must be dispensed in special child proof containers.

2. Education of the parent regarding:
   a. Potential household poisons.
   b. Supervision of toddler’s behavior.
   c. Information’s on poisons & their management.

**Short notes about specific poisonings**

1/ Kerosene poisoning:

- The most common form poisoning in this country while probably the rarest in other parts of the world where kerosene is not used at home. The lower viscosity is responsible for frequent aspiration pneumonia, after absorption from GI tract it causes CNS depression. Both induction of vomiting & gastric lavage are contraindicated in the treatment of all hydrocarbons, charcoal is not effective, the use of steroid & antibiotics is controversial.

- If large quantities have been consumed & life is in danger aspiration of the stomach through an NGT, provided a cuffed ETT secured the air ways may be lifesaving. In general hydrocarbon poisoning may result in addition to inhalation to cardiac, neurological, GI, pulmonary, renal, hepatic, metabolic & hematological manifestations.
2/ Paracetamol (acetaminophen) poisoning

- The most serious effect of which is hepatic toxicity especially if more than 150mg/kg had been ingested, even 75mg/kg might be damaging liver in children who are under weight (for any cause) & those receiving enzyme inducers as carbamazepine, phenytoin & rifampicin.

- The clinical course of severe paracetamol poisoning includes;
  - **Day 1**: GI symptoms
  - **Day 2**: rising serum bilirubin, SGOT, SGPT & PT
  - **Day 3, 4 & 5**: peak hepatotoxicity
  - **Day 6 & 7**: possible recovery in non-fatal cases.

- Always give activated charcoal within the 1st hour of poisoning & in severe poisoning consider N-acetylcysteine (NAC) 2 hours after the administration of charcoal {to minimize the adsorbent effect on (NAC)} or better if given by infusion in 5% dextrose.

3/ Tricyclic antidepressants:

- Harmful effects of which include:
  - Cardiac effects: widened QRS, P-R & QT intervals, atrio-ventricular heart block, atrial & ventricular arrhythmias, hypo or hypertension.
  - Anticholinergic effects: convulsion, coma, tachycardia, worm, flushed & dry skin, decreased bowel sounds, urinary retention & dilated pupils.
  - CNS effects: agitation, ataxia & hallucination.

- The most common causes of death in this poisoning are hypotension & cardiac arrhythmias.

- Management steps include:
  - Gastric lavage.
  - Charcoal.
  - Treat convulsion with valium, phenobarbitone or phenytoin
  - Treat effects with:
    - Induce alkalemia by hyperventilation & NaHCO₃
    - Lidocaine 1mg/kg
    - Phenytoin infusion over 30 min.
    - Defibrillation
    - Normal saline -+ presser agents for hypotension

4/ Iron poisoning:

- Toxic effects of iron include direct corrosive effects on GIT & systemic effects on CVS, metabolic, CNS & frequently hepatic failure. Ingestion of less than 40mg/kg of elemental iron is considered nontoxic, abdominal x-ray may show iron tablets.

- 4 phases of clinical picture are known;
  - **Phase 1**: 1st 6 hours; abdominal pain, vomiting, bloody diarrhea & drowsiness.
  - **Phase 2**: 6-24 hours; apparent recovery.
  - **Phase 3**: 24-48 hours; fever, shock, metabolic acidosis, convulsion, coma & liver affection.
  - **Phase 4**: 4-6 weeks after ingestion; GIT stricture & obstruction & hepatotoxicity.

- Management;
  - GI decontamination.
  - General supportive measure.
  - Specific chelation by deferoxamine.
  - Dialysis.
  - Exchange transfusion may be considered in extremely severe poisoning especially in young children.
5/ Caustic agent poisoning:

- Most of the household cleaning products contain caustic agents.
- Caustic agents include acids & alkalis, both of which have damaging effect on the tissues on contact.
- Acids cause damage by coagulation necrosis, a process that limits further penetration of acid in to deeper tissues, while alkalis cause liquefaction necrosis, which is why alkali can penetrate tissues producing further damage. The extent of tissue injury depends on the PH of the ingested substance (severe damage occurs if PH is more than 12.5), its concentration & the period of contact with the tissue.
- Clinically patient with caustic ingestion may show drooling, vomiting, stridor, dysphagia, oral & perioral erythema, oral & pharyngeal ulceration.
- Management principles include:
  - For acids:
    - Don’t induce vomiting.
    - Don’t give HCO\(_3\) to neutralize.
    - Don’t give charcoal (useless).
    - Irrigate all contaminated areas with copious amount of water.
    - Oral dilute milk or water for ingested poisons.
    - Use of systemic steroid is controversial.
    - Esophagoscopy to evaluate caustic effect.
    - Treatment of esophageal strictures by repeated mechanical dilatation, placement of stents & in severe cases resection of the strictured area.

6/ Salicylate poisoning:

- Ingestion of more than 150mg/kg will cause toxic symptoms as initial respiratory alkalosis which is soon followed by metabolic acidosis, ketosis, hypoglycemia, vomiting, diarrhea, sweating, fever, dehydration, tinnitus, vertigo, restlessness, confusion, convulsions, coma & circulatory collapse.
- Treatment includes:
  - Gastric lavage for up to 4 hours after ingestion.
  - Activated charcoal 1mg/kg.
  - I.V. fluid & electrolytes.
  - Vitamin K to correct hypoprothrombinemia.
  - Severe bleeding may necessitate blood, FFP & clotting factors.
  - Forced alkaline diuresis.
  - Hemodialysis.

7/ CO poisoning:

- CO poisoning results from inhalation of fire smoke, automobile exhaust & also from ingestion of paint removers.
- CO results in cellular hypoxia by 3 means;
  1. Preferential binding to Hb at the site of O\(_2\).
  2. Shifting O2 dissociation curve to the left & by this decreasing O\(_2\) delivery to the tissues.
  3. Binding to cytochrome oxidase & affecting intracellular O\(_2\) transport.
- In addition to cellular hypoxia CO poisoning may damage other tissues & organs e.g. the respiratory system is affected by damage to the mucociliary function, bronchospasm & pulmonary edema.
- CNS effects include: amnesia, slurred speech, headache, convolution, cerebral edema & deafness.
- Cardiac effects include: myocardial necrosis, changing the ECG to range from ST segment abnormalities to atrial fibrillation.
• Affection of muscles results in myoglobinurea & renal failure.
• Skin manifestations include blistering, edema & rarely a characteristic cherry red discoloration.
• 100% O₂ (better if given under 2 atmospheric pressures) is the cornerstone of treatment.
• NOTE: the half-life of CO is 5 hours in room air, 1 hour in 100% FiO₂ while only ½ hour in 100%FiO₂ under pressure.

8/Smoke inhalation:

• Occurs when patients are trapped in closed spaces, during a fire.
• Cellular hypoxia is the main ultimate result.
• Inhalation of very hot gases causes thermal injury to the upper airways resulting in edema & obstruction.
• Chemical injury to the lungs is due to inhalation of toxic products from combustion of PVC, cotton & plastics.
• CO & cyanide are sometimes produced after combustion & cause severe affection of O₂ delivery at tissues. Bronchopneumonia nearly always follows smoke inhalation within 2-3 days.
• Management of smoke inhalation includes ETT, 100% O₂ & cyanide antidote (25% sodium thiosulfate which binds to cyanide to form an easily excretable form, sodium thiocyanide).

9/Lead poisoning:

• The main sources of exposure to lead are: paint of old houses, gasoline, automobile batteries, surma & old printing factories.
• Chronic lead intoxication usually occurs in children with ‘pica’
• Lead encephalopathy may present suddenly with convulsions, raised ICP & coma, or more gradually with drowsiness, ataxia, hyperactivity intellectual impairment.
• Other suggestive features of lead poisoning are:
  o Lines of increased metaphyseal densities of the long bones seen on x-ray.
  o Abdominal x-ray may show radio-opaque shadows.
  o Basophilic stippling of the red cells
  o Resistant iron deficiency anemia.
• Lead is generally deposited in bones.
• Acute infections may mobilize lead from storage areas in bones & cause acute lead encephalopathy.
• Management lines include; removal of the source, decontamination & chelating agent as BAL, EDTA (edathamil), in addition to ICP lowering agents (dexamethasone & mannitol)