بسم الله الرحمن الرحيم

Pediatrics

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POISONING

Poisoning in children is a *common clinical problem* & common cause of hospital admission for observation & treatment. Poisoning can occur with *drugs, cleaning agents, insecticides, paints, petroleum products, plants, & inhalation of toxic gases* as carbon monoxide or other toxic gases or fumes. **Accidental poisoning** is the *most common* cause & it usually occurs in children *below the age of 5 years,* the age at which children usually put things in their mouths. *Family stresses & lack of supervision* are important contributing factors. **Intentional poisoning (child abuse)** should also be considered especially in children below the age of 5 years. *Suicide attempts* may be also considered in adolescents. **Iatrogenic poisoning** due to drug overdose is also common. *Digoxin, theophylline, & anticonvulsants* are the most commonly encountered drugs in iatrogenic poisoning. **Drug abuse** in adolescents as *alcohol, psychostimulants, antidepressants, & hallucinogens* should also be considered. *More than 90% of toxic exposure in children occur in the home,* & most involve only a single substance. Ingestion is the most common route of poisoning exposure (76% of cases), with the dermal, ophthalmic, & inhalation routes each occurring in about 6% of cases. *More than 75% of pediatric poisoning exposures can be managed without direct medical intervention,* because either the product involved is not inherently very toxic or the quantity of the material involved is not sufficient to produce significant toxic effects. Death due to unintentional poisoning in young children is *uncommon* owing to increased product safety measures (e.g., child-resistant packaging), increased poison prevention education, early recognition of exposure, & improvements in medical management.

MANAGEMENT PLAN FOR POISONING

It is important to emphasize that identification of the poison is not the most critical aspect in diagnosis & management & precious time should not be lost trying to identify the causative poison. Good supportive care is the backbone of any successful therapy of poisoned children.

A) History. Obtaining an *accurate problem-oriented history* is of paramount importance if a poisoning has occurred or is suspected. Precise history should include the following :

1. **Description of toxin :** This should include its name, ingredients & their concentrations. Consultation with a *poison control center* can usually provide important help.

2. **Clinical manifestations :** Due to the multiplicity of drugs & other several agents that can cause poisoning, *the pathophysiological changes can almost affect every system.* The clinical manifestations & the severity of findings depend on the *causative agent, amount taken, & time interval between the exposure to poisoning & the onset of medical intervention. Respiratory, cardiovascular, & neurologic* manifestations are the most common, most serious, & the main causes of death in fatal cases. *Gastrointestinal & cutaneous* manifestations are also common presentations. In some cases, the diagnosis of poisoning is clear & obvious when a frank history of drug intake or ingestion of a toxic substances is obtained. In other cases, the diagnosis is difficult or not even considered. Therefore, *the possibility of poisoning should be considered in any child, especially below 5 years, who acutely develops respiratory distress, shock, cardiac arrhythmias, abnormal behaviours, abnormal movements (like ataxia & dystonia), convulsions, disturbed consciousness, severe digestive manifestations, or cutaneous manifestations.*
3. **Magnitude of exposure** (the amount & the concentration of poison, the age & the weight of the child, the length of contact time for inhalation, ocular or dermal exposure).
4. **Time of exposure :** For some product, toxic manifestations may be delayed for hours or days (important in therapy & prognosis).
5. **Progression of symptoms** (important in therapy & prognosis).
6. **Medical history** (underlying diseases, concurrent drug therapy, pregnancy).
7. **Demographic information** (address, telephone No.).

B) Initial medical care

1. At home : If the patient treated at home, *follow-up assessment calls should be made approximately 0.5, 1, & 4 hours after exposure*. Any change in the patient's condition may necessitate admission of the child to *emergency department* at hospital.

2. At hospital : Once the patient has arrived in the appropriate medical care setting, *initial attention should focus on life support,* with primary emphasis on *cardiorespiratory care.* Initial treatment of *shock, dysrhythmias, metabolic* disturbances (temperature abnormalities, acid-base disorders, electrolyte disorders, blood sugar disorders) are extremely important.

C) Investigations & follow-up :

All product containers thought to be related to exposure should be collected & transported with the patient. If the patient has vomited, the emesis should also be brought to the emergency department for possible *toxicologic analysis*. For some intoxications (e.g., salicylates, acetaminophen, iron, methanol, ethylene glycol), *blood levels* are integral to the treatment plan. Comperhensive qualitatitve *"drug screens"* vary widely in their ability to detect toxins & generally *add little informations*(urine is the best sample for drug screens). Repeated assessment of the general condition, vital signs, blood counts, blood gases, serum electrolytes & blood sugar are important.

D) Measures directed to the poison

1. **Antidote therapy :** Unfortunately, *only few drugs & poisons* of which a specific antidote is available. These include *carbon monoxide* (100% O2), *acetaminophen* (N-Acetylcysteine), *cyanide* (cyanide antidote kit : Na nitrate, Na thiosulphate), *organophosphate & carbamate insecticide* (atropine, pralidoxime), *methemoglobinemia* (methylene blue), *opiates & narcotics* (naloxone), *iron* (deferoxamine), *lead* (EDTA, DMSA), *extrapyramidal symptoms* (diphenhydramine), *digoxin* (digoxin-specific Fab antidotes), *anticholinergic agents* (physostigmine) & *benzodiazepines* (flumazenil).
2. **Preventing absorption :** Most toxin are rapidly absorbed from the *gastrointestinal tract* or through *inhalation.* Many may also be well absorbed upon *dermal* contact. Prompt action to remove the toxin & minimize contact with the absorptive surface is crucial & may prevent the development of major toxicity. Dermal & ocular decontamination can be accomplished by flushing the affected area with tepid water. For inhaled toxin, decontamination is generally accomplished by moving the patient to fresh air or, if necessary, administering O2. *Several procedures* are used to prevent absorption of a toxin from the stomach & GIT, & each has *limitations & risks.* In general, most *liquid drug* products are almost completely absorbed within *30 min* of ingestion, & most *solid dosage* forms within *1-2 hr.* Gastrointestinal *decontamination beyond this time is unlikely to be of value*, however, with some poisons , gastric lavage can be done within 12 hr of ingestion (aspirin, opiate, tricyclic antidepressant).
    - ***a)*** **Emesis :** Induction of vomiting by administration of *syrup of ipecac* (10 mL for infants 6-12 mo of age, 15 mL for children 1-12 yr of age, & 30 mL for older children & adults). This is followed by administration of at least 8 oz of water or other clear fluid. The onset of vomiting is usually 20-30 min after dosing in 90-95% of patients. Ipecac *should not be used in infant <6 mo* of age. Because of the delay in onset of emesis & poor yield, *it should not be used as a general treatment for ingestions.* Ipecac induced emesis is *contraindicated after the ingestion of caustic*s, *hydrocarbons,* & agents likely to cause the rapid onset of CNS & cardiovascular symptoms. The use of ipecac syrup has *declined dramatically in the past 2 decades.*
    - **b) Gastric lavage :** This technique involves placing a tube into the stomach to aspirate contents, followed by flushing with aliquots of fluid, usually *normal saline.* Although gastric lavage has been widely used for many years, objective data do not document its efficacy, particularly in children, in whom only small-bore tubes can be used. Lavage is time consuming &, even under the best of circumstances, removes only a fraction of gastric contents. *It should only be used in older children & only in select situations* (e.g., in large doses of aspirin & iron, opiates, tricyclic antidepressants).
    - **c) Activated charcoal :** The use of activated charcoal to prevent absorption of toxins has *increased dramatically in the past 2 decades.* Many, but not all, toxins are adsorbed onto its surface, preventing absorption from the GIT. The usual dose is *10-30 g for a child (1 gm/kg , mixed with water)* & 30-100

g for an adolescent or adult. The dose may be repeated every 2-4 hr until the first charcoal stool appears. About 25% of patients receiving activated charcoal experience one episode of vomiting. Aspiration of activated charcoal into the lungs occasionally occurs.

d) Cathartics : Cathartics are commonly used in conjunction with activated charcoal to hasten the clearance of the charcoal-toxin complex, although no evidence shows that this is of value. Commonly used cathartics are *sorbitol* (maximum dose, 1g/kg), *magnesium sulfate* (maximum dose, 250 mg/kg), & *magnesium citrate* (maximum dose, 250 mL/kg). Cathartics should be used *with care in young children* because of the risk of dehydration & electrolyte imbalance.

e) Whole bowel irrigation : Whole bowel irrigation involves instilling *large volumes of a polyethylene glycol electrolyte solution* (Colyte) into the stomach to cleanse the entire GIT. This technique has been successfully used to remove *slowly absorbed products such as iron or sustained-release preparations.* Whole bowel irrigation can be combined with use of activated charcoal, if appropriate. It should be used with *caution in young children* because of the possibility of fluid & electrolyte imbalance.

3. Enhancing excretion : Enhancing excretion is useful for only a *few toxins.* Dialytic techniques are not useful for drugs that are either highly protein bound or have a large volume of distribution. These techniques are also invasive & associated with risk. Certain procedures can be used for very specific agents.

a) Diuresis : It is a useful technique in drugs mainly excreated by the kidney *(e.g., salicylates & Phenobarbital)* & it is usually combined by *alkalanization* of urine. 2-5 times the maintenance IV fluids are given to establish a urine output of 2-5 ml/kg/hr. Sodium bicarbonate is added to the solution in a concentration of 50-75 mEq/litre. Concomitant use of diuretics as furosemide & mannitol is indicated to ensure high urine flow rate.

b) Dialysis : Hemodialysis & peritoneal dialysis have been used successfully to treat poisonings by select agents *(methanol, ethylene glycol, & large symptomatic ingestions of salicylate or theophylline).*

c) Hemoperfusion : Hemoperfusion is a dialytic technique in which blood is passed through a column of activated charcoal or resin. It has been used successfully to treat large ingestions of *salicylate, theophylline,* & a few other selected agents. It is *rarely used in small children* because of the risks associated with its use.

HYDROCABONS

Hydrocarbons include a wide array of chemical substances found in commercial products. The most important toxic effect of hydrocarbons is *aspiration pneumonitis.* Aspiration usually occurs at the time of ingestion, when coughing & gaging are common,

but can be secondary to vomiting that commonly occur after ingestion. The propensity of hydrocarbon to cause aspiration pneumonitis is *inversely proportional to its viscosity.* Compound hydrocarbons with low viscosity, such as mineral spirits, naphtha, *kerosene, gasoline,* & lamp oil, spread rapidly across surfaces & cover large areas of the lungs when aspirated. Only small quantities *(<1 mL)* of low viscosity hydrocarbons need be aspirated to produce significant injury. Hydrocarbons can be absorbed after *ingestion, inhalation, or dermal* contact. Most hydrocarbons have *anesthetic* properties & can cause transient CNS depression. Aspiration is characterized by coughing, which usually is the first clinical finding. Chest radiograph may be normal for as long as *8-12* hr after aspiration. Respiratory symptoms may remain mild or may rapidly progress to respiratory failure. Fever occurs & may persists for as long as 10 days after aspiration. Accompanying leukocytosis may be misleading because, in most cases of aspiration pneumonitis, no bacteria are present in the lungs. Chest radiographs may remain abnormal long after a patient is clinically normal & should not be used to guide acute treatment. Pneumatoceles may appear on the chest radiograph 2-3 wk after exposure. Several chlorinated solvents, most notably carbon tetrachloride, can produce *hepatic* toxicity.

A few hydrocarbons have also been associated with *renal* toxicity. Benzene is known to cause *cancer* in human after long-term exposure (mostly AML). Nitrobenzene, aniline, & related compounds can produce *methemoglobinemia.* A number of volatile hydrocarbons, including toluene, Propellants, refrigerants, & volatile nitrites, are *commonly abused by inhalation.* Some of these substances can sensitize the myocardium with the risk of *dysrhythmias & sudden death.* Chronic abuse of these agents can lead to cerebral atrophy, neuropsychological changes, peripheral neuropathy, & renal disease. All volatile hydrocarbons are lipid solvents & can cause defatting of skin, producing local irritation or, with prolonged exposure, chemical burns.

Treatment. *Emesis is contraindicated* because of the risk of aspiration. Likewise, *gastric lavage is contraindicated, except under special circumstances*, because of the risk of vomiting & aspiration. Activated charcoal also is not useful, because it does not bind to common hydrocarbons. If hydrocarbon-induced pneumonitis develops , *respiratory treatment is supportive*. *Corticosteroids should be avoided,* because they are not effective & may be harmful. *Prophylactic antibiotics should not be given* because bacterial pneumonia occurs in only very small percentage of cases. Respiratory failure has been successfully trated with both standard ventilation & with extracorporeal membrane oxygenation (ECMO).

ORGANOPHOSPHATES & CARBAMATES

The most commonly used insecticides are either organophosphates or carbamates. Both of these classes are *inhibitors of cholinesterase enzymes.* Nerve agent used in warfare are organophosphates. Most pediatric poisonings occur as the result of accidental exposure to insecticides in & around the home or farm.

Clinical manifestations. Clinical manifestations of organophosphates & carbamates toxicity relate to the accumulation of *acetylcholine* at peripheral nicotinic & muscarinic

synapses & in the CNS. *Muscarinic signs & symptoms* include diaphoresis, emesis, urinary & fecal incontinence, tearing, drooling, bronchorrhea & bronchospasm, miosis, & hypotension & bradycardia. *Nicotinic signs & symotoms* include muscle weakness, fasciculations, tremors, hypoventilation, hypertension, tachycardia & dysrhythmias. *CNS effect* include malaise, confusion, delirium, seizures, & comma. Symptoms caused by carbamate toxicity are usually less severe than those seen with organophosphates. RBC cholinesterase level is decreased.

Treatment. Basic decontamination should be done on exposed persons. *Activated charcoal* can be used for gastric decontamination. *Basic supportive care* should be provided, including fluid & electrolyte replacement & intubation with artificial ventilation if necessary. Tow antidotes are useful in treatment : atropine & pralidoxime. *Atropine,* which block the acetylcholine receptors, is useful for both organophosphates & carbamates. It is most effective at reversing the muscarinic & CNS effects. Often, large doses of atropine must be administered by continuous infusion *(0.05 mg/kg IV repeated q5-10 min as needed.* Dilute in 1-2 mL of NS for ET instillation). *Pralidoxime* chemically breaks the bond between the organophosphates & the enzyme, liberating the enzyme & degrading the organophosphates *(25-50 mg/kg over 5-10 min*, maximum 200mg/min, can be repeated after 1-2 hr then q10-12 hr as needed). Pralidoxime is only effective if used before the bond *"ages"* & becomes permanent ("aging" occurs over the 2-3 days after exposure). Pralidoxime is not necessary for carbamate poisonings because the bond between the insecticide & the enzyme degrades spontaneously. Without treatment, symptoms of organophosphate poisoning may persist for weeks, requiring continous supportive care. Even with treatment, neurologic symptoms may occur & may persist.

CAUSTICS

Caustics include *acids & alkalis* as well as few common *oxidizing agents* such as bleach. Acids coagulate proteins causing *tissue necrosis*. Alkalis digest & dissolve proteins, producing *liquefaction necrosis* with the risk of perforation if the injury is located in the intestinal tract. The severity of the chemical burn produced depends on the pH, the concentration of the agent, & the length of contact time. Agents with a pH <2 or >12 are most likely to produce significant injury.

Clinical manifestations. Ingestion of caustic materials may produce oral burns visualized as reddened areas or whitish plaques. Symptoms include pain, drooling, vomiting, or difficulty or refusal to swallow. Circumferential burns of the esophagus are prone to cause *strictures* on healing which may require repeated dilation or surgical correction. Strong acid may sometimes produce scaring around the pylorus, leading to delayed onset of gastric obstruction. Caustics on the skin or in the eye can cause significant tissue damage.

Treatment. Initial treatment of caustic exposure includes thorough removal of the product from the skin or eye by flushing with water. Contaminated clothing should also be removed. Ingested agents should be rinsed from the oral cavity. *Emesis & lavage are contraindicated.* Activated charcoal should not be used , because it does not bind these

agents. Patients should be evaluated for evidence of esophageal burns &, if symptoms are present, oral fluids or solid should be withheld. The absence of visible oral injury does not preclude significant esophageal lesions. *Endoscopy should be performed in symptomatic patients or those in whom injury is highly suspect on the basis of history.* The use of corticosteroids & esophageal stents is controversial. Prophylactic antibiotics do not improve outcomes.