drug poisoning

(interactive lecture)

poisoning is one of the commonest causes of ICU(PICU) admissions, it is about 3-5% of early admissions, it is not rare we have at least one case every month.
Which age do you think it is the commonest? Almost more than 90% of cases happen before the age of 5 years.

poisoning centers in saudi arabia:
king khalid hospital - king faisal - king fahad - national guard - MOH poisoning center (it serves everyone)-..... etc

-70-80% of child poisoning is below 6 years of age.
- 10-12 years old (adolescents) it's usually suicide attempt rather than poisoning. The difference between poisoning and suicide:

poisoning	suicide
accidental	intentional
very young child (2-3 years)	adolescents (10-12 years old) usually females
one medication	multiple medications

Poisoning in children may be:
• Accidental – the vast majority < 5 years old
• Deliberate self-poisoning in older children
• Non-accidental as a form of child abuse
• Iatrogenic.
Adolescents can be intentional or experimental.

Although many thousands of young children are rushed to doctors' surgeries or hospital for urgent
medical attention following accidental ingestion, most do not develop serious symptoms, as they ingest only

a small quantity of poison or take relatively non-toxic substances. However, a small percentage of
children become seriously ill and a very few children die from poisoning each year.

what are the poisoning agents that available at every home:
medications (paracetamol) - household cleaners (flash - clorox) - insecticide (in the kitchens - gardens) - oral hypoglycemics (23-25% of our population are diabetics) - cardiac medications - cosmetics (makeup) - iron tablets or syrup - TSA - SSRI - OCP(This is common but not serious)…...
there is a misbelief that all medications should be in the refrigerator, it's not only some medications.
We should use a safety cabinet to put all medication away from children.

If you have a call from a relative because his 3years old child (15kg) was next to a bottle of panadol syrup which was empty, there is medication on his hands and mouth. what are you going to tell him:
ABC - wash oral mucosa - (induce vomiting?→ outdated)
take a brief history:
1- **what** is the medication he took?
2- **when** that happened? e.g. if she told you this happened 5-10 days ago then this is not important. If it happened within the last hour then pay close attention to it.
3- **how much** did he take? we can ask about it indirectly, when you opened the bottle? How much was left? ask about the scene and other sites of the home soiled by the syrup like the floor?
We need to have an idea about these medications' volume and concentration (paracetamol syrup is 100 ml), to calculate the concentration of ingested drugs, if it reaches the toxic dose, by

knowing the volume and concentration of the bottle and the ingested amount.
you are required to know the doses of common medications like paracetamol (10-15 ml/ kg). doses in pediatrics are per kg
To know if the child received an acceptable dose or high leads to poisoning you have to calculate based on the weight.
our pt was 15 kg and the mother told you there was about third left in the bottle (which is 100 ml) around 20-30 ml, hands and mouth were soiled so maximum the child took 30 ml, then we have to calculate the concentration, each 5ml contains 125mg so, 10 ml → 250 mg, so 30 ml → 750 mg while the maximum dose for this child 15ml x 15kg = 225 mg so it exceeds the upper limit of acceptable dose by 3x (triple). then we have to calculate the toxic dose in pediatrics 70-80 mg/kg which is very high dose, 150 mg/kg like if he took a full bottle 100ml (100ml has 2250mg= 2250(mg)/15(weight) =150 mg/kg which is very toxic, when in doubt for such doses tell them to come to ER
4- **how's the child doing now since the time of accident**?
you need to know the pt situation if the pt is seizing no benefit from asking the previous questions you have to act. The pt is playing and ok, even though bring him to ER because some medications like paracetamol and iron supplement(early effect is mild abdominal pain but after 8-12 hr present with hepatic involvement, after 24-48 hr fulminant hepatic failure) have a late effect. inappropriate advice may kill so when in doubt ask them to come to ER even if his clinical condition is good and playing, but some medications may have immediate effect like daonil (oral hypoglycemic)10 tablets→ blood glucose 0 within half an hour and start to seize, then you need to instruct the mother to position him to left (to prevent aspiration which can lead to respiratory compromise) and what to do in the way to the nearest medical center.
the patient arrives at the ER, what to do ? ABCDEF.. then

quick brief history and examination (vertex to heel with full exposure) because the pt isn't stable even if he’s stable don’t waste time.
methods of intoxication with insecticide:
inhalation- skin (dermal)- oral- ocular- IV (like in hospitals)
examination :
head: 1-pupil size miosis(organophosphate(insecticide),opioid, pontine hemorrhage -) or mydriasis.
Story: A case I will never forget is a child transferred from the Al-Iman hospital.The child was only 5 months old and they intubated him after he collapsed then sent to a tertiary center. After examination,nothing was found except that he was not able to breathe on his own. A very smart resident found a very small pinpoint pupil which was unusual. In this case there are many differential diagnoses and by going through the history we thought of some ddx?
1-Intracranial bleeding.
2- Medication e.g opioids
3- Organophosphate: Its antidote is atropine.

2- mouth: smell - corrosive agent (chemical-acid) like flash or clorox (flash is more dangerous it may kill because even 5 ml will melt the mouth and esophagus with risk of perforation and stenosis leads to horrible life) What do you think is more serious Clorox or flash? **Flash** will go through oropharynx to the esophagus and
melt the GI so the child will end up having multiple operations. Also, he might collapse and lose his life.
Clorox can be less serious cause it has a very bad smell so the child will take a few mL then through it away.
Even if he took a good amount, this isn’t as serious and we can send him home.
skin examination and wash any substance
vital signs: some medication causes bradycardia like beta blockers

and some can cause tachycardia like salicylate. hyper-hypotension. hypoventilation caused by opioids, hyperventilation like salicylate because it stimulates respiratory center.
Quick investigations(in general not only for paracetamol):
glucocheck→ glucose level (most important) Many medications can cause hypoglycemia and the patient may have convulsions that will not respond to anticonvulsant medication then he will arrest and die
-CBC(anemia with pica) - ABG - U&E (baseline and monitoring)- liver function test (as baseline and monitoring)- renal profile - level of medication (if you know it) - toxicology screen.
Pica is a psychological disorder characterized by an appetite for substances that are largely non-nutritive like ice, clay, soil or paper.
Most of the time there is no witness and the story is unknown or minimal.
DDx of unconscious (comatose) pediatric pt in ER:
trauma - drug poisoning
comatose pt → take them directly to the resuscitation room to stabilize(ABCDEF), put him on cardiorespiratory monitor, IV access(if not available we do intraosseous needle you have to know it), oxygen, airway support (hypoventilating pt will desaturation then have high co2 will accumulate leading to acidosis → more drowsy, more hypotensive and may arrest) If there is visible thing in the mouth remove it, DON'T try to push it down it may block the airway. pt is drowsy for 3-4 hrs → admit to PICU to investigate and do the appropriate intervention if needed.If the pt is blue and not breathing → intubate (mechanical ventilation) don't wait for PICU to do it stat with ambu bag till intubated by expert.
stable pt → stay in ER to plan the management. a decision is made within 2-3 min in ER (quick look and assessment), then pt ready for intervention.
interventions:
1- get rid of the ingested drug by **NG tube**, be careful it has indications and contraindications(coma), pt well, active, no corrosive ingested and normal GCS you can put it.If GCS is 15 normal

3= brain dead
GCS= 7 intubate the child.

there is a reported case of perforation. If the child has corrosive ingestion DON'T put NG tube (absolute contraindication). After NG tube insertion, start gastric washing at the beginning there will be some medication, 30-50 cc of normal saline in and out, and repeat it till the fluid comes out clear at that point you can stop, but don't remove the NG tube (for possible administration of medication).

Indication of GL:
1-Huge amount of medication ingested within the past one hour.
2-Serious or fatal medication like anti hyperglycemic medication.
3-Huge amounts of iron> you need to do the gastric lavage.
Contraindication of GL:
1-More than one hour as there is no benefit for it has already passed to the stomach.
2- Corrosive material (Absolute contraindication) can cause damage to the other normal parts.
3- Change in sensorium: it will cause aspiration no protective airway reflexes.

Usually give 1 g/ kg within 2-3 hours from the poisoning event given (if after 2 hours this doesn't work).This is done by using an NGT if the child is around 2 years. We can also give it mixed with water or Pepsi
if the child is cooperative and older than 5 years.

2- then: get rid of the drug that passed through the antrum to duodenum by **activated charcoal.** its role is decreasing the absorption from intestine by adsorbing (تلتصق)to the drug making the particles larger so can't be absorbed by villi (adsorbs ingested toxins within the gastrointestinal tract preventing the systemic absorption of that toxin). It's not given to pt present after 24hrs of poisoning, it's given **within 4-6hrs** from poisoning and the earlier the better the effect. we give 1gm/kg and we can give it multiple times 2 or3 times. given orally (ex: mixed with juice) in case of co-operative child, or through NG tube if unco-operative child. if pt is fighting there is risk of vomiting and aspiration → activated charcoal in the lung (very difficult)→ severe

ARDS and need intubation, more sequelae may happen but we don't stop giving it because of this risk which is "might happen", but we tell the parents about the complications might happen and that we need to go for it. some areas give (cathetrix"not sure what was the name") which is magnesium sulfate and normal saline → it increases the GI motility and makes the emptying faster, instead of staying 2-3hrs in the gut it stays half an hour.
3-give **antidote**. a lot of medications don't have antidotes like (TCA, hypoglycemic,.....). The antidote for acetaminophen(paracetamol) is N- acetylcysteine. If sure about the type of medication give the antidote and don't wait. we give 17 doses (you don't need to memorize the doses, you can review it when you need it). If you want to know the level of the drug you have to wait for 4-6hrs to be in the circulation with good distribution, but we don't need to wait and lose 6-8 hrs. the result of drug level can be, no poisoning, probable poisoning or definit poisoning, we deal differently with each result. anyway start the antidote(N- acetylcysteine) because its side-effects much better(milder) than the complications of the poisoning.
admit the pt to the ward and monitor if stable but if liver function starts to drop the next day admit to PICU.
and to the PICU if not stable.
4- may need to do dialysis to remove the toxin from the blood(deteriorating pt even after antidote, still having high levels of drug, or not improving),
5-acidification and alkalinization of urine (the drug can pass more easily in alkaline urine than acidic) by giving certain agents.
ALL the steps need to be done (step 4&5 not always) and the antidote depends on the amount ingested and level of the drug.
-in iron poisoning there is a specific situation to confirm iron ingestion → do abdominal X-ray because it's radiopaque.

If we find the tablets in the stomach we can do endoscopy with sedation to remove the tablets one by one, if iron syrup, do the steps like paracetamol. iron antidote is deferoxamine(Desferal)
start it even if in doubt. iron level(TIBC) can be obtained it doesn't long time. In iron poisoning child may have pinkish urine.

induction of vomiting is not done anymore (outdated), but if he vomited spontaneously it's ok.

extra: from illustrated

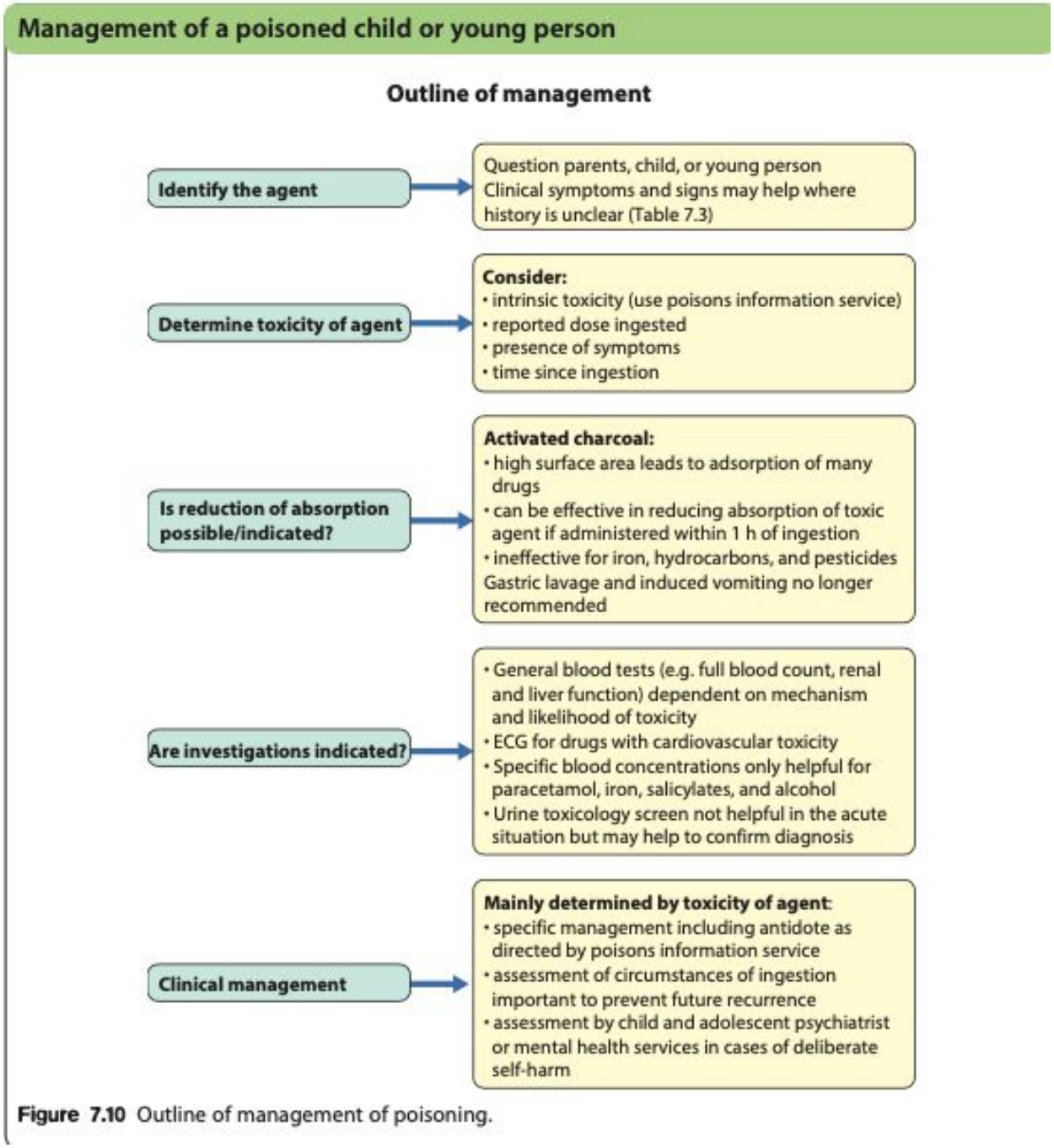

Figure 7.10 Outline of management of poisoning.

Agent	Clinical symptoms	Mechanism	Management
Paracetamol	Early: • abdominal pain, vomiting Later (12 h to 24 h): • liver failure	Initial gastric irritation Toxic metabolite (NAPQI) produced by saturation of liver metabolism	Risk assessed by measuring plasma paracetamol concentration Treat with intravenous acetylcysteine if concentration is high or liver function abnormal
Button batteries	Abdominal pain Gut perforation and stricture formation	Leakage: corrosion of gut wall due to electrical circuit production	X-ray of chest and abdomen to confirm ingestion and identify position Endoscopic removal is recommended if in the oesophagus, the object fails to pass, or symptoms are present (e.g. abdominal pain or melaena)
Carbon monoxide	Early: • headache, nausea Later: • confusion, drowsiness leading to coma	Binds to haemoglobin causing tissue hypoxia	High-flow oxygen to hasten dissociation of carbon monoxide The role of hyperbaric oxygen therapy is unclear
Salicylates	Early: • vomiting, tinnitus Later: • respiratory alkalosis followed by metabolic acidosis	Direct stimulation of respiratory centre Uncouples oxidative phosphorylation leading to metabolic acidosis and hypoglycaemia	Plasma salicylate concentration 2–4 h after ingestion helps to estimate toxicity Alkalinization of urine increases excretion of salicylates. Haemodialysis also effectively removes salicylate
Tricyclic antidepressants	Early: • tachycardia, drowsiness, dry mouth Later: • arrhythmias, seizures	Anti-cholinergic effects, interference with cardiac conduction pathways	Treatment of arrhythmias with sodium bicarbonate Support ventilation
Ethylene glycol (anti-freeze)	Early: • intoxication Later: • tachycardia, metabolic acidosis leading to renal failure	Production of toxic metabolites that interfere with intracellular energy production	Fomepizole inhibits the production of toxic metabolites; alcohol may also be used but has more adverse effects Haemodialysis to remove toxic metabolites in severe cases
Alcohol (accidental or experimenting by older children)	Hypoglycaemia Coma Respiratory failure	Direct inhibitory effect on glycolysis in the liver and neurotransmission in the brain	Monitor blood glucose and correct if necessary. Support ventilation if required Blood alcohol levels may help to predict severity

Agent	Clinical symptoms	Mechanism	Management
Iron	Initial: vomiting, diarrhoea, haematemesis, melaena, acute gastric ulceration Latent period of improvement 6–12 h later: drowsiness, coma, shock, liver failure with hypoglycaemia, and convulsions Long term: gut strictures	Local corrosive effect on gut mucosa Disruption of oxidative phosphorylation in mitochondria leads to free radical production, lipid peroxidation, and metabolic acidosis	Serious toxicity if >75 mg/kg elemental iron ingested Serum iron level 4 h after ingestion is the best laboratory measure of severity Intravenous desferoxamine chelates iron and should be administered in cases of moderate-to-severe toxicity
Hydrocarbons (e.g. paraffin, kerosene)	Pneumonitis Coma	Low viscosity and high volatility makes aspiration easy, resulting in direct lung toxicity Direct inhibitory effect on neurotransmission in the brain	No specific antidote – supportive treatment only
Organophosphorus pesticides	Cholinergic effects: • salivation, lacrimation, urination, diarrhoea and vomiting, muscle weakness, cramps and paralysis, bradycardia. and hypotension Central nervous system effects: • seizures and coma	Inhibition of acetylcholinesterase resulting in accumulation of acetylcholine throughout the nervous system	Supportive care Atropine (often in large doses) as an anticholinergic agent Pralidoxime to reactivate acetylcholinesterase

illustrated is the reference of this lecture

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