

Iron									
Pathophysiology	Direct caustic effect on GI mucosa → hemorrhagic necrosis; multisystem toxicity 2/2 mitochondrial poison; iron absorbed at duodenum/jejunum								
Symptoms	<p><u>If no significant GI symptoms w/i first 6 hrs after overdose, very low likelihood of significant toxicity</u></p> <table border="1"> <tr> <td>Phase I (30min – 6h)</td><td>GI sx: vomiting, diarrhea, GI bleeding</td></tr> <tr> <td>Phase II (6h – 24h)</td><td>Latent period: apparent improvement</td></tr> <tr> <td>Phase III (4h-4days)</td><td> Hepatotoxicity: hepatocellular injury, AG metabolic acidosis (↑ lactic acid), coma, seizures, multi-organ failure, shock Labs: ↑ bili, ↑ LFTs, ↑ glucose, ↑ PT/INR, ↑ BUN </td></tr> <tr> <td>Phase IV (2-8 wks)</td><td>Late effects: possible bowel obstruction</td></tr> </table>	Phase I (30min – 6h)	GI sx: vomiting, diarrhea, GI bleeding	Phase II (6h – 24h)	Latent period: apparent improvement	Phase III (4h-4days)	Hepatotoxicity: hepatocellular injury, AG metabolic acidosis (↑ lactic acid), coma, seizures, multi-organ failure, shock Labs: ↑ bili, ↑ LFTs, ↑ glucose, ↑ PT/INR, ↑ BUN	Phase IV (2-8 wks)	Late effects: possible bowel obstruction
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Evaluation	KUB (radio-opaque pills), Fe level, VBG/ABG, lytes, BUN/Cr, glucose, LFTs, PT/INR, CB								
Management	Support ABC's, replace fluid/blood losses, GI decontamination, IV deferoxamine (severe sx, iron level > 500 mcg/d w/ clinical symptoms, sig AG met acidosis)								

Lead*	
Toxic Dose	No safe lead level exists
Pathophysiology	Interferes w/ interactions of divalent cations and sulfhydryl groups leading to widespread physiologic effects and clinical toxicity.
Symptoms	<ul style="list-style-type: none"> • Lower levels: Abdominal pain, constipation, anorexia, vomiting, dev delays, aggression, hyperactivity • Higher levels: drowsiness, clumsiness, ataxia • Severe levels: decreased consciousness, coma, seizures, death (usually 2/2 cerebral edema)
Evaluation	Lead levels, CBC (microcytic anemia + basophilic stippling of RBC), FEP (free erythrocyte protoporphyrin), BUN/Cr, AST/ALT, x-ray (radio-opaque flecks)
Management	<ul style="list-style-type: none"> • Prevention is key: screening and lead levels at WCC (9-12 mo, 2 years) • Gastric decontamination: whole bowel irrigation • Chelation therapy (depending on lead levels) • See: https://www.cdc.gov/nceh/lead/acclpp/actions_blls.html • Seminal Article: CDC. Managing elevated blood lead levels among young children: Recommendations from the Advisory Committee on Childhood Lead Poisoning Prevention, Atlanta: CDC; 2002 • BCH has a separate Environmental Health clinic and service that can assist w/ management

Drugs of Abuse	
Ethanol	
Hx/PE	Euphoria, loss of coordination, ataxia, slurred speech, nystagmus, nausea, vomiting, hypoglycemia (especially in young children), seizures, coma, respiratory depression
Dx	Blood ethanol level, D-stick
Management	Supportive; secure airway if unresponsive, no gag reflex

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