

Welcome to OHSU talk shorts. Obtain ion concentration, check acid base status and look for visible iron pills on. If concentration is 500 micrograms per deciliter or greater, treatment with deferoxamine and began whole bowel irrigation. If a patient reports iron ingestion and at any time develops metabolic acidosis or hemodynamic instability with hypotension and tachycardia, send serum iron concentration and begin deferoxamine infusion with whole bowel irrigation and disposition to the ICU. Profound toxicity develops from hypovolemia vasodilation, poor cardiac output with decreased tissue perfusion, resulting in a worsening metabolic acidosis. A dose of 20 to 40mg/kg can cause GI symptoms like abdominal pain, nausea, vomiting, and diarrhea, potentially even some hematemesis. While practice patterns vary in general for ion concentrations over 500 micrograms per deciliter, deferoxamine chelation is. Note that a fourhour iron level will give you a steady state of concentration. However, for the sick patient, don't wait for hours to obtain this laboratory test for the ill appearing patient with reported ingestion of a toxic dose of iron, particularly the patient with hypotension and metabolic acidosis. Because the vomiting patient does not tolerate charcoal well, it is probably best to avoid this therapy as. Whole bowel irrigation can be used by installing polyethylene glycol through a nasogastric tube. Dehydration contributes to ill appearance and hypertension in the iron poisoned patient with significant GI symptoms, including hematemesis or hematochezia. The patient can develop anion gap acidosis, hypoglycemia due to sympathomimetic effects, and margination leukocytosis from a stress state. Patients can develop lethargy and coma, worsening GI bleed, acute kidney injury, and pulmonary edema. Stage five, which rarely manifests in patients, can include gastric scarring, pyloric stenosis, and small bowel stricture. Note that iron carbonyl and iron polysaccharide contained in many of the newer iron formulations do not typically cause toxicity. I've deferoxamine can cause hypertension, so this infusion will be increased over time and titrated to the ability of the patient's blood pressure to tolerate infusion. Ongoing cellular toxicity due to iron's effect at blocking oxidative phosphorylation continues. On her arrival, you note she is tearful, she's actively vomiting, and you note some blood tinge to her emesis. This can develop from Mallory Weiss tears or from true mucosal irritation. First, you should provide IV fluid resuscitation through two large bore peripheral IVs. Iron is not absorbed by activated charcoal in the stomach, so it is not generally recommended, although some providers have used charcoal in serious cases. Acknowledging that the pH in the duodenum may make adsorption of iron to activated charcoal more successful. The patient developed some worsening hypertension in the ED, so it was continued on IV fluids and started on norepinephrine. The color of her urine, initially rose color had become clear, norepinephrine was weaned off overnight, and her deferoxamine infusion was dropped the next day. Let's review the iron treatment algorithm for the asymptomatic patient with a history of small ingestion of under 20mg/kg. The patient can be disposed home or to a psychiatric care facility, as proper for ingestion of more than 20mg/kg or if GI symptoms are present. Because its sulfate has an S, we divide it by five to obtain the iron content per tablet. She's a 60-kilogram patient, so her dose is approximately 100mg/kg. A dose of 60mg/kg can cause more serious effects like hypotension, tachycardia. Anion gap acidosis, hyperglycemia, and leukocytosis. In serious ingestion, such as this one, the infusion may be slowed to 5mg/kg per hour as needed. You might consider treating with norepinephrine if hypotension is not responsive to fluids or is worsening. So, if you're starting a deferoxamine infusion in the emergency department, consider

.reminding your inpatient colleagues about this