

a 17-year-old female is in or out with hematemesis after ingesting a bottle of her aunt's iron pills. She was after dispositioned to an inpatient psychiatric facility. Profound toxicity develops from hypovolemia vasodilation, poor cardiac output with decreased tissue perfusion, resulting in a worsening metabolic acidosis. Iron is not absorbed by activated charcoal in the stomach, so it is not generally recommended. Whole bowel irrigation can be used by installing polyethylene glycol. While practice patterns vary in general for iron concentrations over 500 micrograms per deciliter, deferoxamine chelation is. Note that a four-hour iron level will give you a steady state of concentration. Late phase one signs are hypotension, tachycardia, and The patient can develop anion gap acidosis, hypoglycemia, and margination leukocytosis. 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. Ongoing cellular toxicity due to iron's effect at blocking oxidative phosphorylation continues. Because its sulfate has an S She's a 60-kilogram patient, so her dose is approximately 100mg/kg. This can develop from Mallory Weiss tears or from true mucosal irritation. The patient developed some worsening hypertension in the Ed, so it was continued on IV fluids and started on norepinephrine. Within 16 hours, the patient had resolution of her hypertension. norepinephrine was weaned off overnight, and her deferoxamine infusion was dropped the next day. A dose of 60mg/kg can cause 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. I've deferoxamine can cause hypertension