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CASE QUIZ

E.P.C.M.S.

Four Year Old Boy with Tablet Ingestion

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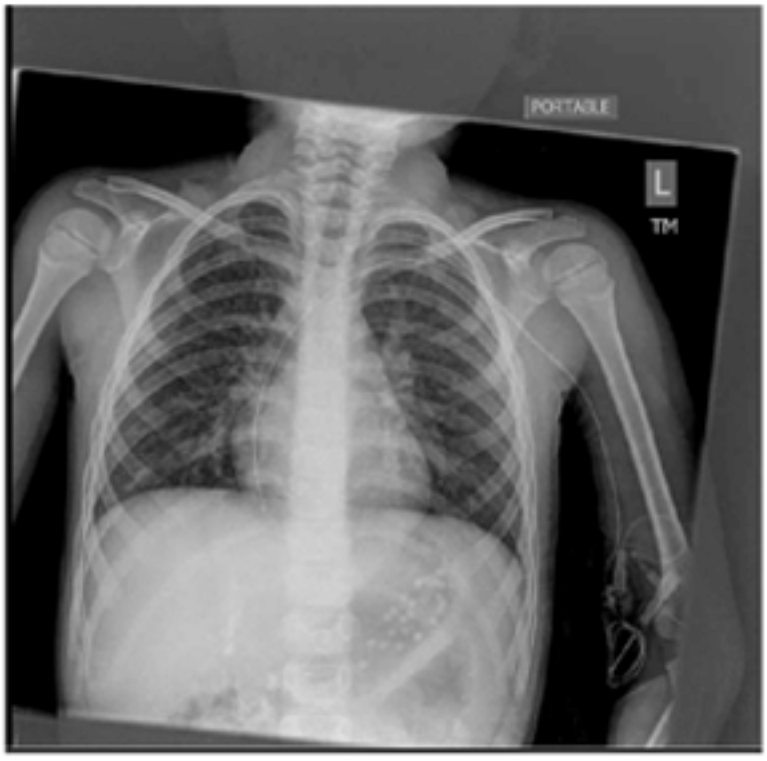
You are called to consult on a 4-year-old 20-kg child who has attention-deficit disorder and was transferred to your hospital approximately 6 hours ago following the ingestion of his mother's prenatal vitamins. His mother believes that he ingested at least 30 tablets containing 325 mg ferrous sulfate, which is 20% elemental iron. The child has been vomiting frequently despite intravenous maintenance hydration. On physical examination, the afebrile child's heart rate is 150 beats/min, blood pressure is 74/50 mm Hg, and respiratory rate is 22 breaths/min. Abdominal radiography has been obtained (**Figure**).



Of the following, in addition to transferring the child to the PICU, the next MOST important intervention is

A. activated charcoal treatment
B. antiemetic administration
C. intravenous deferoxamine infusion
D. normal saline infusion
E. whole bowel irrigation

Answer on page 10

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CASE QUIZ

Four Year Old Boy with Tablet Ingestion (Continued)

D: The boy described in the vignette is believed to have ingested 325-mg tablets of ferrous sulfate, which contain 20% elemental iron. Thus, each tablet contains 65 mg of elemental iron. If he ingested 30 tablets, the total elemental iron ingestion is 1,950 mg (30 X 65 mg), which is 97.5 mg/kg for this 20-kg boy. The radiograph reveals the pills in his stomach. For any suspected iron ingestion, the clinician always must ask what form of iron was ingested because the toxic dose depends on the amount of elemental iron ingested. Ferrous gluconate is 12% elemental iron, ferrous sulfate is 20%, and ferrous fumarate is 33%. Minimum and lethal doses have not been firmly established, but the following tenets apply: 1) Patients who ingest less than 20 mg/kg of elemental iron usually are asymptomatic, 2) Ingestions of 20 to 60 mg/kg may produce symptoms of toxicity, and 3) Ingestions of more than 60 mg/kg can be associated with serious toxicity. Accordingly, the boy in the vignette is at risk for serious toxicity.

Iron is toxic to many cells (heart, liver, central nervous system), damaging tissue through free radical production and lipid peroxidation. javascript:showModal('/subscriber/modal/march/march-q2-content-specifications') Iron causes mucosal cell necrosis, impairment of capillary permeability, alteration of the lipid membrane of the mitochondria, uncoupling of oxidative phosphorylation, inhibition of enzymatic processes in the Krebs cycle, direct vasodilatation, and inhibition of serum proteases (eg, thrombin). The direct myocardial effect, vasodilatation, gastrointestinal fluid losses, and metabolic acidosis can lead to the development of shock, cardiovascular collapse, and death. Because this boy is vomiting and hypotensive, the most immediate concerns are assessment of airway, breathing, and circulation and aggressive fluid resuscitation with normal saline.

Under normal conditions, approximately 10% of ingested iron is absorbed from the intestine and bound to transferrin, using only 15% to 35% of the binding capacity of transferrin. The total iron-binding capacity is a crude measure of the ability of serum proteins, including transferrin, to bind iron and ranges from 300 to 400 mcg/dL. It is higher than the serum iron concentration because of a low degree of saturation. However, when iron concentrations rise following an overdose, transferrin becomes saturated, allowing excess iron to circulate freely in the serum and cause toxicities. Ingestions of more than 60 mg/kg correlate with serum iron concentrations greater than 500 mcg/dL (89.5 mcmol/L).

Iron toxicity may be categorized into five phases:

Phase 1: Effects of iron on the gastrointestinal system, with vomiting and diarrhea, which may turn bloody, and may last several hours

Phase 2: Apparent recovery, which may last 24 to 48 hours

Phase 3: Recurrence of gastrointestinal symptoms, lethargy, coma, acidosis, coagulopathy, renal failure, cardiovascular collapse, elevation of liver function tests, and worsening acidosis

Phase 4: Fulminant hepatic failure, occurring 2 to 5 days following ingestion (dose-dependent, rare, and usually fatal)

Phase 5: Gastrointestinal healing, with pyloric and bowel scarring, occurring 2 weeks after ingestion

Following assessment of airway, breathing, and circulation, serum iron concentrations should be assessed 3 to 5 hours after ingestion. This is the most useful laboratory value to obtain. Peak iron values of less than 350 mcg/dL (62.7 mcmol/L) are associated with minimal toxicity, 350 to 500 mcg/dL (62.7 to 89.5 mcmol/L) with moderate toxicity, and more than 500 mcg/dL (89.5mc mol/L) with potentially severe toxicity. Because iron is cleared rapidly from the blood to the liver, serum iron values may be deceptively low if measured late. An abdominal radiograph should be obtained because the presence of tablets correlates with the severity of the ingestion. Following stabilization, the goal is to remove the iron from the gastrointestinal tract. There is currently no available evidence to support the use of gastric lavage in iron overdose. In addition, iron binds poorly to activated charcoal. However, whole bowel irrigation with a polyethylene glycol electrolyte lavage solution is beneficial. The solution is administered orally or through a nasogastric tube at 20 to 40 mL/kg per hour in children and 1.5 to 2.0 L for adolescents and young adults. It is continued until the rectal effluent is clear. Hemodialysis and hemoperfusion are not effective because iron has a large volume of distribution.

Chelation therapy should be considered for patients who have severe symptoms (altered mental status, hemodynamic instability, persistent vomiting, bloody diarrhea), anion gap acidosis, serum iron concentrations greater than 500 mcg/dL (89.5 mcmol/L), and a significant number of pills on radiograph. Deferoxamine chelates iron to form a water-soluble compound that is excreted renally. It has a short half-life and must be administered as a continuous intravenous infusion beginning at 15 mg/kg per hour for up to 24 hours (the maximum rate of infusion is 35 mg/kg per hour). Rapid administration leads to hypotension. If serum iron concentrations are not available, elevated serum glucose values and leukocyte counts are 100% predictive of serum iron concentrations greater than 300 mcg/mL (53.7 mcmol/L).

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