Acute Fulminant Liver Failure Due To Iron Intoxication Requiring Emergent Liver Transplantation

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Introduction

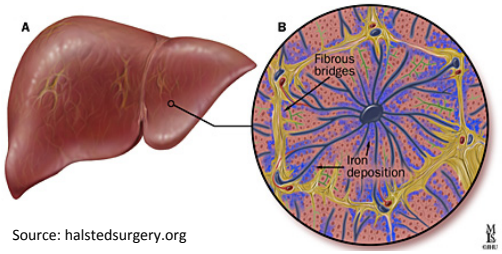
The majority of iron toxicity cases occur in children as a result of accidental ingestion, but can also be due to intentional overdose. Iron toxicity can have damaging effects on the heart, endocrine glands, liver, and other organ systems. Although the initial symptoms are mild, such as abdominal pain, vomiting, and diarrhea, it can quickly progress to hypotension, shock and end-organ failure.

Case Description

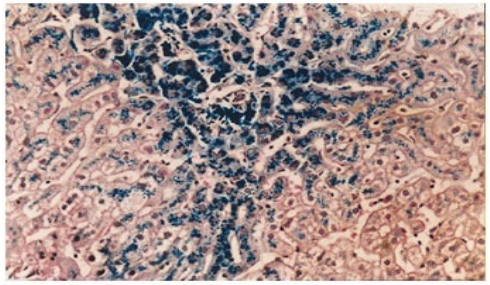
- 14-year-old girl with a PMH of mild asthma, transferred from outside hospital for acute fulminant hepatic failure from iron ingestion.
- Patient had ingested multiple tablets of ferrous sulfate, amoxicillin, and ibuprofen.
- Brought to ED after developing emesis, diarrhea, tonic-clonic movements, and altered mental status. Toxicology screen was negative.
- Labs were notable for acidosis with **pH 7.18**, **WBC 43.5**, and **lactate 6.7**, and empiric antibiotics were started.
- **Transaminases 4000s**, **elevated bilirubin**, and **INR 5.0**.
- She was started on N-acetylcysteine and deferoxamine after discussion with poison control center and transferred to CUMC PICU for further monitoring.
- PICU course complicated by altered mental status requiring intubation, and hematemesis and hematochezia with hypotension requiring massive transfusion. She was also started on rifaximin and lactulose for hepatic encephalopathy.
- Emergently listed as **status 1A for liver transplant** and underwent an orthotopic liver transplant on day 6 after ingestion.
- Intraoperative course was significant for minimal blood loss requiring 2 units of pRBC, 4 units of FFP and 1 unit of cryoprecipitate. She was on low dose epinephrine and vasopressin drips after reperfusion, which were discontinued on arrival to the PICU. Her mental status improved and she was extubated on POD #2. She was discharged on POD #15 to home with prolonged psychiatric support.

Stages of Iron Poisoning

Stage	Time post-ingestion	Description
1	Within 6h	Vomiting, hematemesis, explosive diarrhea, irritability, abdominal pain, lethargy
2	6-48h	Up to 24h of apparent improvement (latent period)
3	12-24h	Shock, seizures, fever, coagulopathy, metabolic acidosis
4	2-5 days	Liver failure, jaundice, coagulopathy, hypoglycemia
5	2-5 weeks	Gastric outlet/duodenal obstruction due to scarring



Source: halstedsurgery.org



Deposits of hemosiderin in the hepatocytes, especially in periportal areas

Source: NEJM 1998;339:269-270.

Discussion

Hepatotoxicity from iron poisoning is uncommon, making it low on differential diagnoses. However, when it does occur, it is associated with a high mortality rate. Damage from iron occurs in the periportal regions where cells receive blood with high oxygen and iron, resulting in free radical formation leading to damage. The periportal region is responsible for hepatic regeneration, so hepatic damage due to iron poisoning will carry a much worse prognosis compared to intoxication from other drugs. In the first 6 hrs, patients typically present with vomiting, diarrhea, and lethargy. It can progress quickly to hypotension, metabolic acidosis, coagulopathy and shock. Liver failure occurs after a day, although the progression varies case by case. Like our patient, upper and lower GI bleeding can also occur from coagulopathies. The specific treatment is IV deferoxamine, an iron chelator, but the only definitive treatment is emergent liver transplantation. It is important for physicians to recognize the acuity of the injury and the emergent need for liver transplantation. Another important and easily forgotten step is calling poison control center to report findings and discuss options.

References

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